The Journal of Thoracic Surgery

VOL. 14 DECEMBER, 1945 NO. 6

Original Communications

TRAUMATIC WET LUNG

OBSERVATIONS ON CERTAIN PHYSIOLOGIC FUNDAMENTALS OF THORACIC TRAUMA

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NOTABLE strides in the understanding of thoracic physiology were made during the war of 1914 to 1918. The brilliant studies of Graham and Bell[1] on pneumothorax did much to put thoracic surgery on a firm physiologic footing. Their proper emphasis on the mediastinum and on the all-important relationship between open pneumothorax and respiratory reserve, and the practical application of these fundamentals to the empyema problem made medical history. These facts are too well known to warrant review. So well have these principles been disseminated, and so well indoctrinated has this generation of physicians become, that in a large experience it is a thing of extreme rarity to see an open pneumothorax that has not been recognized and well managed. Likewise the closed or tension pneumothorax is a problem to which practically all are alert and instances in which the patient dies because of an unrecognized or untreated tension pneumothorax are gratifyingly few and far between. Conscientious and painstaking cognizance of the "pleural" and "mediastinal" factors concerned in chest trauma has paid tremendous dividends in lives saved and pulmonary crippling prevented.

Significant studies were stimulated by the mass trauma in World War I in still another thoracic realm. Due largely to the emphasis of Pasteur[2] of England the entity of "acute massive collapse" of the lung was recognized, and its relation to trauma stressed. During the early postwar period surgeons were prolific in their postulates concerning the nature and etiology of this interesting and serious phenomenon. Elliott and Dingley[3] suggested a reflex arrest of the homolateral hemidiaphragm with retention of bronchial secretions as the causative factor. Briscoe[4] was also convinced of the relationship of an immobile diaphragm but hypothecated diaphragmatic infection as the reason for the immobility of that structure. Bradford[5] postulated a reflex bronchiolar spasm with spasmodic occlusion of the bronchioles in the involved segment. Churchill[6] accepted the explanation of Elliott and Dingley and revived the term atelectasis which had been more or less forgotten by surgeons. A vastly improved understanding of the nature of posttraumatic and postoperative pulmonary complications was to be the direct outgrowth of these studies.

Received for publication Feb. 5, 1945.

The experience of World War II should do much to extend and integrate these brilliant beginnings. This study is an attempt to emphasize what is felt to be a most important aspect of the problem of thoracic trauma.

During the past ten months, as members of Thoracic Surgical Teams of an Auxiliary Surgical Group, we have studied and treated approximately 1,500 patients with thoracic trauma. This study has led us to certain conclusions which we believe to be of the greatest importance in the management of chest trauma. All types and degrees of trauma to the thorax and its contents were observed, varying from simple uncomplicated thoracic wall contusions to the most severe types of penetrating and perforating wounds. The majority of these patients were first seen more than seventy-two hours after injury. One hundred fourteen patients were seen within seventy-two hours of the time of injury. Of these, seventy-nine were seen within forty-eight hours of injury, the majority of the seventy-nine within twenty-four hours. It is this early group from which the following observations were made.

In this experience two fundamental physiopathologic phenomena have been observed:

1. That in all wounds of the chest to a greater or lesser degree, depending upon the type and severity of the lesion, the lung tissue reacts to produce more than its normal amount of interstitial and intra-alveolar fluid.

2. That in all wounds of the chest the bronchopulmonary tree not only has more fluid to rid itself of, but becomes less capable of doing so. The result of these two phenomena is what may be called for the want of a better term the "wet lung" of trauma. This wet lung is the one common denominator of all significant thoracic trauma. In the one case it may be so slight as to be of no consequence. In many it is the factor upon which the outcome will depend. Failure to take cognizance of this fact will frequently lead to death or to the establishment of chronic pulmonary invalidism. Practically all cases of thoracic trauma have some degree of pulmonary "wetness." This may be transitory or so slight as to be almost unrecognizable. In a significant number of cases it was present to a strikingly more severe degree than would be anticipated from the relative nonseverity of the initial lesion.

The well-developed early case of traumatic wet lung presents the following clinical features, during what may be termed the "primary" phase of the syndrome:

The patient is often apprehensive. Dyspnea of some degree is always present and is accompanied by paroxysms of a painful cough which, while usually productive, is not effective in the sense that it is emptying the bronchopulmonary segments. This is an important distinction. The cough has an unmistakably moist element, even after the patient has raised a small amount of sputum. Because of the pain element, respirations are frequently grunting in nature. This type of respiration is often referred to loosely as "diaphragmatic."

On physical examination of the chest, if there is no significant associated pulmonary or pleural pathology such as hemothorax or pulmonary hematoma, the percussion note and tactile fremitus are not changed significantly. There is, however, restriction of motion of the involved side with reduction in intensity

of breath sounds. The outstanding feature is the presence of râles, often on both sides, although usually more marked on the involved side. The râles are bronchial in character and consist of wheezes and rhonchi. When marked, these are palpable and may even be audible without the aid of a stethescope. They are not infrequently mistaken for friction rubs. Some of these patients appear to be having a typical attack of bronchial asthma. In many cases the râles are predominantly moist or even bubbling in character, and so profuse as to be identical with the type heard in the long familiar, simple, nontraumatic pulmonary edema. Diffuse moist râles are especially characteristic of the primary phase of traumatic wet lung. All gradations are encountered. Obviously, associated lesions may greatly alter, modify, or even mask these signs and symptoms. The coexistent hemothorax, pneumothorax, or other lesion may divert the physician's attention. Since the râles are often present even over the uninvolved side, this may be a valuable clue that a significant degree of wet lung exists or is developing. X-ray studies may show lesions of the ribs, pleura, or lungs. Again x-ray findings may be completely absent even in the presence of marked auscultatory moisture.

It cannot be overemphasized that the lung reacts to trauma just as uniquely as the brain or any highly specialized organ. This fundamental response results in increased fluid production and retention of that fluid in the bronchopulmonary system. It would be fallacious on the basis of the evidence at present to formulate too precisely a concept explaining this all-important substratum of thoracic trauma. It is tempting at this point to postulate the existence of reflex bronchial and bronchiolar spasm originating from a painful chest wall or parietal pleura and to attempt a corollary with the recent experimental work of DeTakats and associates.[7] These workers claim to have demonstrated reflex bronchospasm in the cat. Such an hypothesis would explain some of the phenomena observed in traumatic wet lung, yet would fail of complete physiologic consistency. In fact, the whole case for bronchospasm rests upon evidence that is far from being unquestioned. A consideration of the incontrovertible aspects of this syndrome emphasizes the role of chest wall pain as a dominant factor in its inception, regardless of the mechanism of the pain influence. Pain, which is an almost invariable component of chest trauma, indisputably hampers the cough mechanism to a serious degree. This emphasizes, of course, the important role played by the thoracic cage wall in traumatic wet lung.

Regardless of the ultimate explanation of the cause or causes, the entire mechanism by which the lung might be able to cope with the primary lesion, whether that be a laceration, a pulmonary hematoma, or a retained shell fragment, is deranged. That mechanism must be restored if the lung is to react favorably to surgery or other therapy. If the aberration is marked, the mechanism must be restored if the patient is to survive.

More and more clinical evidence accrues, as our experience grows in handling thoracic cases both in the forward hospitals and in the base hospitals, that a definite "one-two" relationship may exist between the "wet lung" of trauma as described and the massive collapse of Pasteur.[2] It is a highly attractive hypothesis that this "wet" phase is the antecedent stage, and in a sense the precipitating event, of the "silent" massive collapse. Such an assumption would correlate nicely with the universal observation that massive collapse is virtually unheard of in this theater among those who are alert to the prompt

recognition and treatment of traumatic "wet lung." That it leads in many cases, if untreated, to less marked atelectases is of course unquestioned.

Recognition of wet lung in no wise detracts from the importance of other lesions. Principles laid down for their therapy are rational and they are in the main well recognized and well managed. It is, however, stressed that this all-important common denominator of thoracic trauma exists and must be recognized. Once it is realized that the traumatized thorax, be it from the slightest chest wall contusion to the severest penetrating type of wound, is reacting fundamentally in a way peculiar to itself, and that these changes will profoundly influence all concomitant lesions, all prognosis, and all therapy, we shall be well on the way to a more rational approach to the whole problem of thoracic trauma.

All too frequently, however, the primary thoracic lesion masks this underlying physiopathologic substratum. There is something inherently frightening about respiratory distress both to patient and to doctor. Perhaps this is why the efforts for its relief are all too frequently as frantic and as ineffectual as the patient's efforts to get his breath. Measures which relieve the thoracic wall pain and result in an effective cough, thereby clearing the obstructed bronchopulmonary tree, will usually change the picture completely. One can then see the primary lesion for what it is. The blood in the pleura, the shattered ribs, or the shell fragment in the lung will loom less ominous and, perhaps, less urgent. At least the lung has been aided to regain the ability to cope with whatever it must face, either from the surgeon or from the injury. The patient will have been prepared for careful, contemplative evaluation, without which definitive therapy, particularly surgery, cannot hope to succeed.

In the therapy of this condition, all procedures must be formulated with the idea of, first, controlling the production of moisture and, second, promoting adequate bronchial drainage. This necessitates the physiologic readjustment of those factors which are primarily responsible for initiating this syndrome. Some of these are vague at present and little understood. Pain originating in the traumatized thoracic cage structures emerges as the most important factor in the development of the unfavorable cycle of events. It is this pain that interferes so seriously with an effective cough mechanism. It is the pain complex, then, that must be combated.

The time-honored method of controlling thoracic wall pain has been adhesive strapping and the generous use of morphine. Both are physiologically unsound approaches and both are ineffectual. While morphine, by raising the pain threshold, makes the patient less aware of pain, it actually diminishes the cough reflex and does not aid in the establishment of a painless cough mechanism. By dulling the sensorium it makes the patient less aware of his own difficulties and therefore an easier victim of them. When adhesive strapping is used, the existing anoxia is further increased by the limitation of chest wall motion imposed by this method. In addition, it is an actual handicap to the propulsive mechanism of cough. Actually it is not infrequent to see patients benefited simply by the removal of adhesive strapping.

The logical therapy, of course, suggests itself. Abolition of pain and physiologic remobilization of the chest wall are the sine qua non of a successful therapeutic effort. Temporary intercostal nerve block was first suggested by Latteri.[8] Unfortunately, he suggested the use of alcohol. Later Harmon and co-workers,[9] Rovenstine and Byrd,[10] and others reported striking and lasting relief of pain by procaine block in cases of rib fracture. Unfortunately, these observers misplaced the emphasis and others have been slow to realize the value of their contribution.

In this series injection of procaine has been used either at the site of injury, by intercostal nerve injection, or by thoracic paravertebral sympathetic nerve block with gratifying results. Blocking out the pain stimuli produces immediate and often dramatic results. The cough becomes painless and effective, and evacuation of the bronchopulmonary tree becomes established. The patient will often pass from an alarming state to one of comparative comfort and safety within a very few minutes. While the abolition of pain stimuli from the thoracic cage with the reinstitution of a painless, effective cough mechanism is the sheet anchor of the therapeutic scheme for traumatic wet lung, other forms of therapy will often be necessary.

Some of these patients will be so flooded with bronchopulmonary secretions and so exhausted by their trauma that mechanical evacuation of the tree is urgently indicated. This is most expeditiously accomplished by tracheobronchial catheter aspiration. In a few, bronchoscopic evacuation may be necessary. Aspiration is particularly necessary in those cases complicated by an existing communication between the tracheobronchial tree and the pleural cavity containing fluid.

A certain small group will present evidence of persistent moisture after nerve block and aspiration. In these the administration of atropine may be of value. Intravenous administration is preferred. In some patients psychic factors are very prominent, and before complete control can be achieved, ample sedation must be administered. Once pain has been controlled and an efficient cough mechanism re-established, it is imperative that someone stand by and make the patient cough effectively. In the more resistant cases, where other methods have failed to control the moisture, oxygen delivered at a positive pressure of from 6 to 8 cm. of water is of benefit.

A series of representative cases of traumatic wet lung is reported here. They have been chosen in order to illustrate that this syndrome may occur in a wide variety of thoracic lesions. They include cases of simple and complicated rib fractures and examples of very extensive lesions of the pleura and parenchyma. They have also been selected because they illustrate the principles of therapy outlined.

CASE REPORTS

CASE 1.—A soldier was injured in a steep fall, Sept. 27, 1943. He was admitted to the hospital several hours later complaining of severe pain in the lower left side of the chest. At this time he was raising small amounts of bloodstreaked sputum. On examination he showed moderate dyspnea and he had a painful paroxysmal cough. There was moderate tenderness over the lower half of the left side of the chest. There were no significant changes in tactile fremitus, percussion resonance, or the character of the breath sounds. There were diffuse wheezes and rhonchi bilaterally so that he appeared to be having a typical attack of bronchial asthma. X-ray did not reveal any fractured ribs or any demonstrable parenchymal or pleural lesions.

A procaine intercostal nerve block of the lower six ribs was done. Relief of pain occurred in a very few minutes. With the relief of pain, cough became easy and productive of relatively large amounts of mucoid sputum; by the end of thirty minutes the râles had completely disappeared.

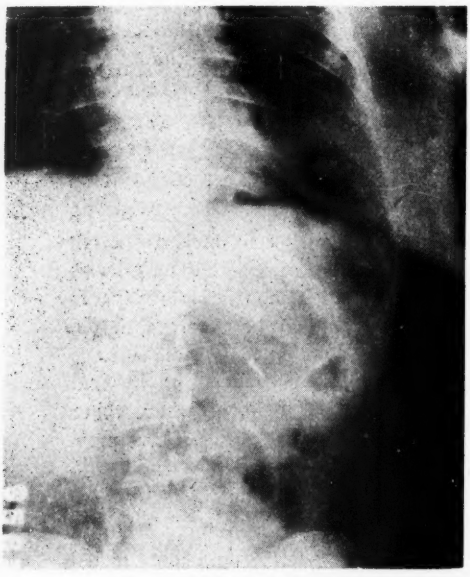
This was a case of simple uncomplicated contusion of the chest wall. Complete relief was obtained by the use of procaine intercostal nerve block.

CASE 2.—A soldier was injured in a truck accident, Feb. 10, 1944, at 8 P.M. He was seen at 6:30 A.M., February 11. At this time he had a painful wet cough, numerous moist râles and wheezes bilaterally, and moderate dyspnea. He had a hematoma of the left side of

the chest wall and a slight amount of subcutaneous emphysema. X-ray films showed fractures of the ninth, tenth, and eleventh ribs on the left in the posterior axillary line (Fig. 1). There was no demonstrable parenchymal or pleural pathology.

A procaine intercostal nerve block of the lower six ribs on the left side resulted in immediate and complete relief with disappearance of all evidence of pulmonary wetness. No further treatment was required.

This case illustrates the very frequent association of the traumatic wet lung with severe rib fracture, and is illustrative of the prompt and complete relief obtained by intercostal nerve block.



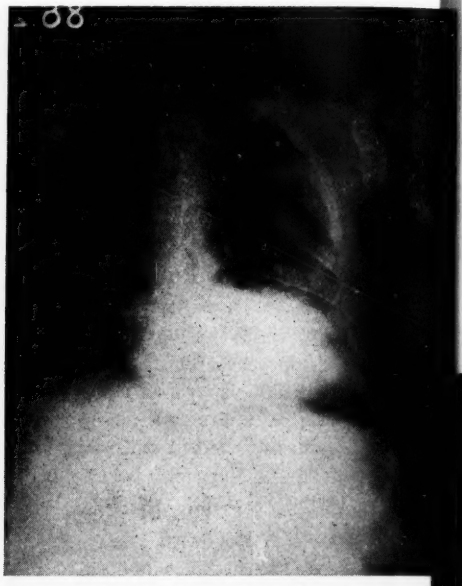


Fig. 1 (Case 2).—Fractures of the ninth, tenth, and eleventh ribs in the posterior axillary line.

Fig. 2. (Case 3).—Anterior and posterior fractures of the left sixth and seventh ribs, producing a "flail" segment in the thoracic wall.

CASE 3.—A patient was injured when he fell off a motorcycle, Feb. 1, 1944, at 2 P.M. When seen at 4 P.M., he had severe pain in the left side of the chest with a paroxysmal, wet, ineffectual cough. There was marked tenderness over the left fifth and sixth ribs with a "flail" segment in this area, which was undergoing paradoxical oscillations. Numerous wheezes and rhonchi were present bilaterally. Dyspnea was moderately intense. X-ray revealed anterior and posterior fractures of the left fifth and sixth ribs (Fig. 2).

A procaine intercostal block of the left third to ninth ribs inclusive resulted in only partial relief of pain. This was supplemented by thoracic paravertebral procaine block of the sympathetic ganglia of the same segments. Following this, there was relief of pain and the paradoxical oscillations of the "flail" segment ceased. Although there was definite improvement in the pulmonary wetness, some moisture persisted. Therefore, $\frac{1}{150}$ gr. of atropine sulfate was administered intravenously; within five minutes the remaining wheezes and rhonchi disappeared and the breathing was entirely normal. The relief of pain lasted for three days, at the end of which time there was a recurrence of pain and "wetness" to a moderate degree. Paravertebral block was repeated with immediate and complete relief.

This case illustrates several features which have been discussed. Chief among these is the fact that in some cases it is necessary to add atropine to the therapeutic regimen before all moisture is controlled. Another feature of this case is that it illustrates the remarkable effect of nerve block on a "flail chest" of limited extent.

CASE 4.—A patient sustained a penetrating wound of the anterior left side of the chest at 1:30 A.M., Feb. 3, 1944. He was admitted at 9 P.M. on February 4 with hemoptysis, dyspnea, painful wet cough, and pain in the liver, left side of the chest, and left shoulder. In the shock tent, his condition was considered to be critical. He was cyanotic and very dyspneic and there were showers of moist râles throughout both lungs. In short, he presented the picture of "pulmonary edema." Tenderness and rigidity were also revealed in the left upper quadrant of the abdomen. X-ray films (Fig. 3) revealed extensive disease in the left lung and left pleural cavity as well as a retained missile in the region of the left diaphragm.

At 10 P.M. on February 4, a procaine intercostal block of the left fourth to ninth ribs was done, and then 200 c.c. of air and blood were aspirated from the left pleural cavity. Following this, there was definite improvement in the pain, dyspnea, cyanosis, and pulmonary moisture. At the same time, the left upper quadrant became soft and remained soft throughout the subsequent course. At 12 P.M. he had a recurrence of the pulmonary difficulties and at this time a tracheal catheter was inserted. A large amount of blood and mucopurulent sputum was aspirated and then a paravertebral sympathetic block of the fourth to ninth dorsal lumbar vertebrae was performed. These procedures resulted in complete relief of pain and very marked regression of the pulmonary moisture. On February 5, this soldier had slight pain in the left side of the chest, associated with moderate dyspnea. Procaine intercostal block of the left fifth to ninth ribs produced immediate and lasting relief.

This patient appeared to be in danger of early death when first seen. Although nerve block aided considerably at first, he later appeared to be completely exhausted and so mechanical aspiration of the tracheobronchus was utilized. Three hours after he was first seen, his condition was improved to the point where the thoracic wound could be débrided. One week after admission he had improved sufficiently to be evacuated safely to a hospital for the definitive therapy of the primary lesions.

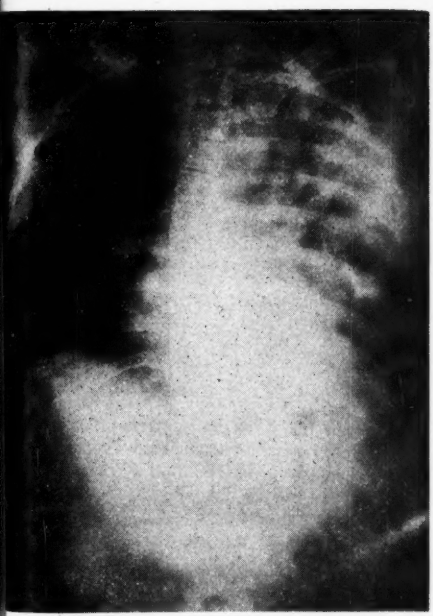


Fig. 3 (Case 4).—Extensive left-sided pulmonary and pleural obscuration, with two retained intrathoracic missiles.

Fig. 4 (Case 5).—A relatively small degree of disease visible; the patient, however, still had an advanced degree of wet lung.

CASE 5.—A soldier sustained a penetrating wound of the left side of the chest at 8 A.M., Feb. 3, 1944. At 10 A.M. on February 4, a 2.5 cm. wound in the left seventh interspace was explored. The missile, which had pierced the diaphragm and grooved the capsule of the spleen, was found in the pleural cavity. Five hundred cubic centimeters of blood were aspirated and the diaphragm was repaired. At 7 P.M. that day, the patient developed signs and symp-

toms of a wet lung. Procaine intercostal nerve block of the left second to tenth ribs succeeded in controlling the pain and relieving the dyspnea and moisture. The next day, February 5, the patient had a recurrence of this difficulty and again nerve block (of the left fifth to ninth ribs) was effective. Following this, however, his condition gradually became worse and by February 7, he was having almost constant paroxysms of wet, ineffectual cough during which he raised only small amounts of white, frothy, mucoid sputum. Dyspnea was marked but he had no pain. He was extremely apprehensive and very restless, appearing to be almost exhausted. An intravenous injection of morphine sulfate, gr. ⅙, and atropine sulfate, gr. 1/150, was given and ten minutes later he was sleeping. A regimen of nembutal, gr. 1½ every eight hours for four doses, was instituted. With this, apprehension and restlessness were allayed and he was able to raise large amounts of thin, mucoid sputum. There was no further recurrence of difficulty (Fig. 4).

This case illustrates that a certain percentage of these patients will require a combination of the therapeutic measures outlined, before the wet lung can be brought under control. While nerve block was valuable in abolishing the thoracic cage pain component of the syndrome, moisture persisted, necessitating the use of atropine. Because of the patient's psychic instability, it was further necessary to quiet him adequately before full therapeutic control was obtained.

CASE 6.*—A soldier was wounded 7 P.M., Jan. 23, 1944, and sustained bilateral sucking wounds, bilateral hemopneumothorax, and bilateral intrapulmonary retained missiles. When admitted at 9:50 P.M. his pulse was 140 per minute and the blood pressure was not obtainable. Immediate therapy consisted of two units of plasma, 500 c.c. of blood, morphine sulfate, gr. ¼, intravenously, and continuous nasal oxygen. At 11:15 P.M., the pulse was 132 per minute and the blood pressure 92/70 mm. Hg. However, at 2:30 A.M. on January 24, the patient became cyanotic and comatose and developed an extreme degree of wet lung. A tracheal catheter was inserted and large amounts of mucoid sputum were aspirated. The pulse improved, cyanosis disappeared, and the patient regained consciousness. The catheter was left in the trachea and intermittent suction applied until 3 P.M., at which time the blood pressure was constant at 108/74 mm. Hg, and the tracheobronchial tree was dry. Both wounds were débrided at this time. Thereafter, at irregular intervals for the next week, aspiration was repeated to treat recurring attacks of wet lung. When admitted to our service, Feb. 5, 1944, he was in good condition (Fig. 5).

In this case, pain was a minimal feature. The tracheobronchial tree was overwhelmed and it filled beyond the point where cough could empty it. Mechanical aid was essential to prevent the patient from drowning in his own secretions. So dire was his plight, in fact, that the highly unusual circumstance was encountered in which the tracheobronchial catheter had to be employed in an indwelling fashion to save the patient's life.

CASE 7.—A soldier was admitted with a traumatic amputation of the right foot Jan. 10, 1944. January 15, he developed cyanosis and moderate dyspnea. For the next few days, he had irregular periods of hyperpnea and there occurred signs of consolidation at the angle of the right scapula. In spite of adequate sulfadiazine and oxygen therapy, his condition became worse and he developed severe pain in the right axilla and extreme cyanosis, on January 26. Physical examination revealed bronchial breath sounds in the right chest laterally and moist râles and rhonchi bilaterally.

At 7 P.M. a procaine intercostal nerve block of the right fourth to ninth ribs gave immediate relief of pain. The breathing became deeper and the cyanosis diminished considerably. By 10 P.M. he was breathing well and he had an effective cough with the production of dark, bloody sputum. He had no pain and the contralateral râles had cleared. X-ray film prior to therapy (Fig. 6) revealed pulmonary infarction.

This patient with pulmonary infarction obtained complete relief of pain by the prompt institution of adequate bronchopulmonary drainage with complete clearing of the wet phenomena by intercostal nerve block.

*We are indebted to Major Lyman A. Brewer, III, for permission to include this case in our series.

DIFFERENTIAL DIAGNOSIS

Traumatic wet lung will have to be distinguished from bronchial asthma, pulmonary edema of cardiac origin, pulmonary edema seen in peripheral vascular failure, and from blast lung.

The history of trauma and the absence of any history of asthmatic attacks will very quickly rule out bronchial asthma.

Since the young soldier is almost never a cardiac subject the problem of differentiating the wet lung of trauma from pulmonary edema of cardiac origin is extremely rare. However, on occasion patients with pulmonary lesions have had such vigorous intravenous fluid therapy that the blood volume becomes too great for an already embarrassed respiratory mechanism and right heart failure supervenes. This picture may be interwoven with that of the traumatic wet lung, each condition intensifying the other.

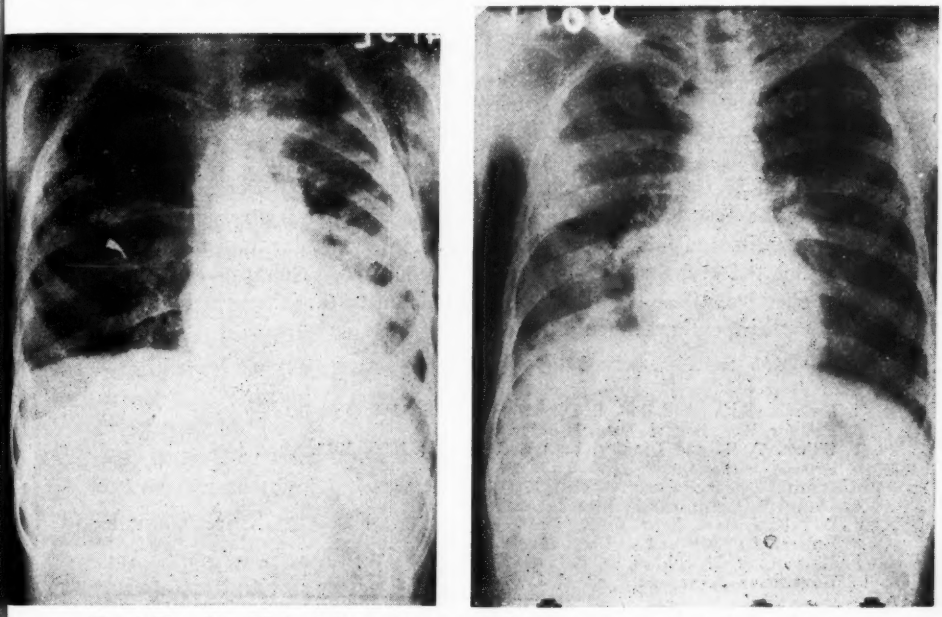


Fig. 5 (Case 6).—Bilateral intrapulmonary shell fragments; one week after injury, extensive left-sided intrapulmonary shadows and subcutaneous emphysema still present.

Fig. 6 (Case 7).—Patchy obscuration on the right, due to multiple right-sided pulmonary infarcts.

Pulmonary edema associated with peripheral vascular failure from either an overwhelming toxemia or the shock of severe trauma will be obvious. The nature of the exciting lesion, pallor, low blood pressure, tachycardia, weak thready pulse, and absence of any significant respiratory difficulty present an entirely different clinical picture than that seen in the wet lung.

In this series of cases, "blast" lung has not been an important problem. Two of the criteria that are said to be essential for the establishment of this diagnosis are missing in the majority of the cases in this series. These are the absence of any significant external evidence of injury to the chest and the presence of hemorrhagic lesions in both lungs. However, it is obvious that many of the features, both physiopathologic and clinical, of wet lung and blast lung are similar. This is to be expected if our concept is rational. Blast lung repre-

sents pulmonary damage in response to a severe blow on the thoracic cage by a wave of highly compressed air. It produces hemorrhage in the parenchyma, pleura, and even the thoracic wall tissues. With this, there develops some degree of pulmonary moisture. This is borne out by a review of the pathologic reports which stress the appearance of pulmonary edema in these cases. It is our belief that blast lung is a severe degree of pulmonary contusion and that the supervening pulmonary edema is traumatic in origin and not the result of right heart failure because of increased resistance in the pulmonary circuit. Indeed, one of the puzzles has been that death in blast lung may occur in the presence of rather minor pulmonary involvement. It would seem then that the principles of therapy outlined here might well aid in the better control of blast lung. If the pulmonary moisture is reduced by nerve block, atropine, tracheobronchial catheterization, or therapeutic oxygen delivered under positive pressure, the patient has a far better chance of recovering from the purely hemorrhagic lesions resulting from the contusion.

SUMMARY AND CONCLUSIONS

An attempt has been made in this paper to focus attention on what is believed to be a most important concept in the management of thoracic trauma. That concept is that the thoracic structures react in a very fundamental way to trauma. This basic physiopathologic response results in a clinical picture we have called by the descriptive term, traumatic wet lung. The interrelationship between thoracic wall, lung, and tracheobronchial tree is stressed. The fundamental features of increased interstitial and intra-alveolar fluid production with bronchial and bronchiolar retention are emphasized. Therapy has been gratifyingly effective. Case records are submitted illustrating the principles discussed and the therapeutic regimen followed.

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THE USE AND CONTROL OF THORACIC SURGICAL TEAMS OF AN AUXILIARY SURGICAL GROUP

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AN AUXILIARY surgical group composed of a group headquarters, general surgical, orthopedic, neurosurgical, maxillofacial, and thoracic surgical teams plus shock, miscellaneous, gas, and dental prosthetic teams has been employed in the American Army for the first time during World War II. It is a theater reserve unit. This organization of surgical and allied teams under one central control has produced a high level of competence as well as economy in the deployment of specialized surgical skill and talent. The remarkable advances which have been made in thoracic surgery since World War I demand that this additional knowledge be applied in the care of the casualties of World War II. It is unique that thoracic surgical teams are organized and functioning as specialty teams for the first time in our Army. All of the other types of surgical teams in the group were employed in World War I. This paper is based on an experience of eighteen months in the employment of an auxiliary surgical group in an active theater of operations. One of the most interesting phases of this experience has been the use and control of the thoracic surgical teams which have been charged with the care of over 2,000 patients suffering thoracic injuries, the vast majority of which involved intrapleural lesions. It is the purpose of this paper to discuss the requirements for team members, functions of the teams, their employment in various hospitals, and the control of the activities of these as well as all other teams of the group.

The thoracic surgical teams are composed of a thoracic surgeon, an assistant surgeon, an anesthetist, a surgical operating room nurse, and two enlisted surgical technicians. The professional qualifications for the head of the team are exacting. He must have a thorough knowledge of the surgical physiology of the thorax, be a competent operator, be a good teacher, and will best be prepared to do his work if he has had thorough training in general surgery as the basis for his training in thoracic surgery. The significance of this requirement is generally appreciated in the peacetime practice of this specialty, but its necessity in war surgery can hardly be overestimated. The assistant surgeon should have had a minimum of two years of surgical training, and a period of several months devoted to the study of the medical aspects of thoracic diseases is very desirable. The requirements in anesthesia are paramount to the success of thoracic surgery, and the severity of the injuries of certain war casualties only emphasizes this requirement. Expertness in the employment of endotracheal and positive pressure anesthesia, bronchoscopic aspiration of the tracheobronchial secretions, the management of shock, a thorough knowledge of the methods of recognizing and correcting disturbed cardiorespiratory physiology, and the sharing of the meticulous pre-and postoperative care of the patient with the surgeon are the minimum requirements for an anesthetist on a thoracic surgical team of an auxiliary surgical group.

Received for publication Feb. 10, 1945.

A surgical nurse on one of these teams has ably described the requirements for this position as follows: "She must not only be a good surgical nurse but in addition must have detailed knowledge of the technical features of the specialty and possess the temperament and adaptability to frequent operative ordeals during long periods of sustained tension."[1] To facilitate her work she must be an able instructor in operating room technique, capable of teaching the principles of asepsis to soldiers who have had no previous experience. The surgical technicians must be good soldiers, well disciplined and demonstrating a tenacity for hard work. Youth, the average intelligence of a high school graduate, inquisitiveness as to the feats of modern surgery, and capability of being inspired by their officers to achieve a high degree of proficiency in the preparation and sterilization of surgical supplies together with a gentleness which will enable them, if required, to be good assistants at the operating table are the prerequisites for good surgical technicians. An individual's qualifications as a truck driver, electrician, mechanic, tent pitcher, and improvisor of operating room fixtures as well as comforts for living under field conditions should be of the highest order to afford the maximum efficiency of the team. Teamwork is essential to success. All members of the team must have the physical stamina which permits frequent and long hours of sustained meticulous work. Fearlessness, keenness of observation, a capacity for rendering immediate correct decisive action, embraced by a sincerity and tenacity of purpose coupled with the delight of adventure are attributes that all team members find enhance the happiness in their work. There is no place for the unstable, unpredictable individual on surgical teams of an auxiliary surgical group.

An intangible quality second only to the professional requirements of team members is their ability to adapt themselves to varied and changing situations.

The teams work in many different hospitals supplementing the regular staff. In the first priority surgical hospital they are charged with the responsibility of the professional service. In other installations they augment the regular surgical staff. In all instances they must adapt themselves cheerfully and readily to the local requirements, facilities, and policies of that institution. Tact should be a natural attribute or one which is rapidly acquired. Applied diplomacy in a true and practical sense pays remarkable dividends and is indispensable to the orderly efficient function of the team. The chronic fault-finder, overflowing with "helpful" suggestions, finds life on a surgical team disappointing. Such individuals are likewise an acute detriment to all other members of the team or teams.

The equipment of a thoracic surgical team is minimal consistent with the performance of its functions. Each team has an army basic instrument set and supplemental thoracic set. The latter includes a bronchoscope and accessories needed for therapeutic bronchoscopic aspirations. An electric suction apparatus and a portable anesthesia apparatus complete the list of integral team equipment. This equipment has been severely tested as to its durability by frequent and repeated sterilization of instruments, rapid moves by motor over difficult terrain, and by moves over air and water routes. It has withstood these tests in admirable fashion. Tentage for quarters and motor transportation are pooled with other teams to assure economy in its use. The personal equipment includes the standard army field equipment. A sleeping bag and an air mattress are cherished comforts.

The function of the thoracic surgical teams as well as all other teams of the group is to augment the staff of hospitals throughout the theater when

the requirements for surgery exceed the capacity of the regular staff. They have functioned in installations as far forward as divisional clearing stations and as far to the rear as base general hospitals in the communication zone. They have also accompanied the assault forces on amphibious landings. They have functioned in all the hospitals in two American Armies and a separate corps, many hospitals in four different base sections, British casualty clearing stations, and British general hospitals. From this experience, certain general principles referable to their functions are applicable:

1. Divisional clearing stations are not designed to provide the hospital facilities nesessary for the proper care of first priority surgical cases and are, therefore, unsuitable installations for the employment of surgical teams.

2. The team should become an integral part of the staff during its temporary stay in any hospital. It should take its turn in operative surgery on the same schedule as the regular surgical staff.

3. The head of the team should be responsible for the preoperative preparation, determination of when to operate, and then the postoperative care and designation of when evacuation of his patients is feasible.

4. Teams should be relieved and return to their own group headquarters as soon as the flow of casualties is decreased to the point that they can be efficiently cared for by the regular staff of the hospital. It is very desirable that teams function in many different hospitals in order that all teams may be able to function efficiently on short notice in any hospital.

5. During inactive or quiet periods at the front, the experience of the individual surgeon should be augmented by duty in a base hospital.

6. The unique experience of the thoracic surgical teams in having the opportunity to be responsible for the professional service in a chest center established in fixed hospitals in North Africa and Italy has proved highly successful. Such a center has permitted the continuity of treatment of a large number of patients who have received their initial surgery by surgeons of the same auxiliary surgical group. A detailed follow-up study of many problems of traumatic thoracic surgery has been accomplished and the contributions therefrom will stand as significant advances in this field of surgery.

7. The maintenance of accurate individual case records is a requirement for the proper function of all teams.

The employment of thoracic surgical teams may be divided into employment in forward areas and in base section areas. In the forward area they form part of the Army medical service and in the base area they are a part of the medical service of the communication zone. In this theater a very highly satisfactory system for caring for the small percentage of most severely wounded has been developed. This system utilizes platoons of a field hospital as small forward surgical hospitals for the care of first priority surgical patients. These are nontransportable casualties and are defined as those who cannot be transported further to the rear without surgery. When field hospitals are used in this manner they are under corps control and they must be somewhat altered in their integral organization and equipment. Additional transportation is also required.

A field hospital employed similar to a station hospital in a base section area or in the vicinity of an air field is staffed and equipped to care for approximately 380 patients, each of its three platoons being capable of the independent operation of a hospital of approximately 100 bed capacity. When employed as a first priority mobile surgical hospital the bed capacity must be

markedly reduced. A patient census in excess of thirty-five patients places an almost insurmountable task on the nursing staff in the postoperative care of the patients and seldom has it been necessary to exceed this bed capacity. It was recognized at the outset that the small staff on the professional service of the hospital was inadequate to render the proper surgical care to these casualties. The surgical teams of an auxiliary surgical group have proved ideal as a source for this needed qualified surgical personnel and these teams have been charged with the surgical care of the wounded treated in these first priority surgical hospitals. The regular hospital staff is fully occupied with the operation of the hospital and with aiding in the management of shock therapy.

The surgical care of casualties, other than first-aid measures and the administration of plasma, is inaugurated at these forward surgical hospitals. They are located in the rear of the divisional area and are in physical conjunction with the division clearing station. The latter installation is the key to triage from the battlefield. Nontransportable casualties are immediately transferred by hand litter to the platoon of the field hospital. "No pause is required for resuscitation or interference with splinting or dressings. Expert surgical management that embraces resuscitation, operation, and prolonged postoperative care becomes immediately available. Cases with a continuing source of shock that cannot be made transportable without an operation are thus salvaged and the desperately wounded receive expert care as far forward as it can be provided."[2] These platoons are capable of moving on very short notice and on arrival at their new location can set up and start functioning in two or three hours. In practice, however, the platoons "leap frog" one another and the platoon or a part of it which is left behind becomes a "holding unit." Sufficient personnel remain with the holding unit to administer postoperative care; this usually includes one surgical team. When all of the patients have been evacuated, in from two to three or even ten days, this unit is available to "leap frog" another platoon and become the forward hospital.

The physical setup of this forward hospital is interesting and has been largely standardized. It is a hospital so erected in ward tents that it forms a cross. The receiving and shock ward is situated at the main entrance with the postoperative ward directly opposite; the x-ray equipment and laboratory occupy a ward tent, and opposite is the operating room. A pyramidal tent forms the central point in the cross. This compact arrangement minimizes the moving of patients, concentrates supplies, simplifies nursing care, decreases heating difficulties during the winter, and provides complete blackout.

It is in this forward surgical hospital that surgical teams have been extensively employed. An active platoon requires four general surgical, one thoracic, and one shock team. The function of the thoracic surgical team is not limited to operative surgery. In fact, much of the work of the thoracic surgeon has been in the capacity of advising in the management of thoracic wounds. He has been instrumental in teaching bronchoscopy to anesthetists and surgeons. His interest in intercostal nerve block in the therapy of thoracic trauma, pre- and postoperative thoracentesis, endotracheal and positive pressure anesthesia, pulmonary re-expansion after surgery, catheter aspiration of tracheobronchial secretions, the management of intrathoracic foreign bodies, and traumatic hemothorax has been a stimulus to all surgeons treating severe thoracic wounds. These facts do not imply that the thoracic surgical team

does little operative surgery, for the converse is quite true. It does mean that with expert opinion in this specialty available at this most forward surgical hospital, that the highest degree of proficiency in the surgical management of these injuries is being attained. It is agreed that certain types of penetrating and perforating wounds, which earlier in our experience were considered as first priority surgical problems, are no longer classified as such and the judicious application of certain therapeutic measures previously enumerated, applied in conjunction with proper shock management, renders these patients transportable, and occasionally the necessity for surgical intervention is obviated. The comparatively large number of severe thoracic injuries which does require first priority surgery has likewise surprised both general and thoracic surgeons. In an unselected series of patients treated during a seven-week period by one thoracic surgical team in a platoon of a field hospital, ninety-two had intrathoracic wounds requiring surgery. Of this number, twenty were combined thoraco-abdominal wounds, sixteen necessitated formal thoracotomy, and the remaining fifty-six were penetrating or perforating injuries. During the same period the team operated upon forty-four patients with abdominal wounds.

It must be repeatedly emphasized that first priority surgical casualties represent only a very small percentage of the total number triaged at the division clearing station. Approximately 8 per cent or one-thirteenth received at this station belong in this category. The great bulk of the wounded is evacuated to evacuation hospitals a few miles to the rear. Likewise, emphasis is warranted in indicating that urgency of the wound and condition of these nontransportable casualties are desperate. Shock of a profound state, that is, systolic blood pressure of 60 mm. of Hg or less, is present in approximately 50 per cent of all first priority surgical cases. Resuscitation measures are heroic and the determination of the proper time to operate requires masterful surgical judgment. The mortality rate in a well-run first priority surgical hospital is high and an over-all rate of 25 per cent is not unusual. A rate of less than 12 to 20 per cent strongly suggests an improper selection of cases for treatment at this installation.[3] To accept other than first priority surgical cases defeats the primary purpose for which such a hospital is designed.

The over-all mortality rate in a carefully studied and unselected series of 346 intra-abdominal wounds, 20 per cent of which were combined thoraco-abdominal wounds treated by surgeons of this group, was 30.6 per cent. Ninety-three per cent of the cases were followed in other installations, evacuation or fixed base hospitals. The primary cause of death in 27 per cent was pulmonary complication, while peritonitis accounted for only 11.4 per cent of the deaths.[4] In another series of 226 intrathoracic wounds, including ninety-seven or 43 per cent thoraco-abdominal injuries, the over-all mortality was 28 per cent.[5] These statistical data are of significance because they demonstrate several important features relative to the functions of thoracic surgical teams in this forward surgical hospital:

1. That at this level in the chain of evacuation the segregation of patients into the fields of surgical specialties is impractical. Approximately 85 per cent of the patients at admissions are those wounded by high explosive missils, as artillery, mortar, mines, and antipersonnel bombs. These agents are notorious for their infliction of multiple wounds and approximately 80 per cent of all nontransportable casualties have multiple major wounds.

2. That thoracic surgical teams working at this level in the chain of evacuation must be competent to do major abdominal surgery, that one team cannot do all of the thoracic surgery required in a first priority surgical hospital, and that the majority of these patients will be treated by general surgical teams. This places an increased responsibility on the thoracic surgeon as it is desirable that he be available for consulting with the general surgeons and that he wisely disseminate his own knowledge in this specialty to all other surgeons. He thus becomes active in the management of approximately 50 per cent of all first priority surgical hospital cases. It has often been found desirable to designate one of the general surgical teams in a platoon whose surgeon is especially interested in thoracic surgery to alternate with the thoracic surgical team in giving first attention to patients suffering urgent chest wounds.

The consulting surgeon of this theater has emphasized that, "A well-run first priority surgical hospital exerts a remarkable favorable influence on the morale of combat troops and their officers. The divisional medical service receives a stimulus to maintain its arduous task by firsthand evidence that the lives of the most desperately wounded may be saved by skillful first-aid measures and rapid evacuation. Splinting is improved, the use of plasma in aid stations is increased, and the temptation for clearing and collecting companies to indulge in heroic surgical procedures for which they were never designed or equipped is removed."[2] The surgical teams of an auxiliary surgical group have had an unusual opportunity in participating in the development of this phase of forward surgery. They have fulfilled their responsibilities in an exemplary manner.

The evacuation hospitals which correspond to the No. 2 hospital in the "three-point forward" system described by Jolly as employed in the Spanish Civil War, usually located a few miles (three to fifteen) to the rear of the first priority surgical hospital, treat approximately 90 per cent of the casualties triaged from the divisional clearing stations. The percentage of abdominal and thoraco-abdominal injuries treated in these installations is low. It is here, however, that the larger number of penetrating and perforating injuries of the thorax, alone or in combination with extremity injuries, are treated. Experience has demonstrated that at this level in the chain of evacuation an appreciable degree of segregation of cases into surgical specialty categories is practical. Thus, the severe head injuries are treated by competent neurosurgeons, maxillofacial wounds by specialists in that field, compound fractures and severe extremity injuries by orthopedic or general surgeons, and thoracic wounds by thoracic surgeons. In such installations, either 400 or 750 bed type, thoracic surgical teams have been particularly useful either to supplement the regular thoracic surgical section of the hospital or to furnish the qualified personnel if there is no experienced thoracic surgeon on the regular hospital staff. Here as in platoons of the field hospital much of the work of the thoracic surgical team concerns not only operative surgery but many nonoperative procedures necessary in the pre- and postoperative management of thoracic wounded. It is not unusual to have a thoracic surgical service in an evacuation hospital of forty patients, for many problems concerning thoracic surgery arise among a large percentage of patients being treated for other injuries in these hospitals. It is highly desirable during the period of peak load of casualties to have two thoracic surgical teams functioning in an evacuation hospital devoting their primary attention to the treatment of wounded whose major injuries involve the thorax.

The extent to which other than urgent intrathoracic surgery is feasible in these hospitals is somewhat dependent upon the tactical situation, the distance and the transportation facilities between the evacuation hospitals, and the fixed hospitals in the base section area. It is a fundamental principle in the operation of all forward hospitals that they must not become engaged in the performance of elective surgery when the need for their bed capacity by battle casualties is imminent. In quiet periods at the front, or when evacuation facilities to the rear are hampered, certain surgical procedures usually reserved for the base section hospitals have been undertaken. These include the removal of intrapulmonary foreign bodies and pulmonary decortication for organizing hemothorax. This was especially true in Tunisia, North Africa, and to some extent in Sicily. During the Italian Campaign after the establishment of general hospitals, these surgical procedures were reserved for general hospitals or designated thoracic surgical centers in fixed hospitals. The experience gained in the management of thoracic wounds by thoracic surgical teams functioning in evacuation hospitals has been another important link affording a broad concept of the over-all surgical problems relating to war wounds of the chest.

In the fixed installations, station or general hospitals in the base section area, a real opportunity has been presented to thoracic surgeons. Fortunately, the teams of an auxiliary surgical group have had the privilege of participating in the development of thoracic surgical centers established in certain fixed hospitals in the base area. The concentration of the special surgical problems involving intrathoracic injuries has permitted vigorous efforts toward solving these problems. The particular problems have evolved about principles of management which are particularly applicable to an overseas theater. Every effort must be made to restore the injured to duty within the theater if this can be accomplished within a designated period, usually ninety days. This policy in its application to intrathoracic injuries has led to several noteworthy contributions. The removal of intrapulmonary foreign bodies within ten days to three weeks after injury has been successfully practiced with patients returning to duty in the theater. "The radical management of massive organizing hemothorax by thoracotomy, evacuation of the clot, and decortication of the lung has proved its effectiveness in returning soldiers to duty and appears to have diminished the incidence of empyema. The same procedure applied to establish post-traumatic empyema with penicillin therapy as an adjunct is followed by immediate healing with a fully expanded lung. It is no longer acceptable to hold that a patient with a penetrating chest wound is making satisfactory progress as long as an empyema has not made itself manifest. The focus has changed from the management of post-traumatic pleural infection to the preservation of lung function. In the history of military surgery this will stand as one of the significant advances of World War II."[2] The thoracic surgical teams of an auxiliary surgical group have been the initial contributors and have successfully exploited these phases of traumatic thoracic surgery. The large number of patients cared for in these centers, the excellent facilities, and the opportunity for careful follow-up study have been valuable to thoracic surgical teams in both forward and rear areas in broadening their knowledge and concepts in the management of thoracic injuries incident to war.

The control of the activities of an auxiliary surgical group is administered by its group headquarters. This headquarters moves with the concentration of teams in a given area. Detachments of a few or several teams are frequently

sent a considerable distance from the headquarters, being temporarily attached to a task force. As soon as the mission for which they were attached is accomplished they return to their own headquarters and are available for duty elsewhere. When the assigned mission is a major one requiring a force the size of an army the entire group is attached to the army. Normally one auxiliary surgical group functions with an army. The coordination of the functions and employment of the group is a responsibility of the group headquarters. This requires the closest liaison between the higher headquarters and in the planning of operations requiring surgical teams. Army and higher headquarters know the tactical situation and plans for future operations. Certain hospitals must frequently be augmented by surgical teams in anticipation of a heavy casualty rate. This is especially true in amphibious landings or in situations in which an army has major forces in separate areas with the intervening territory occupied by the enemy, for example, the Anzio Beachhead.

Explicit cooperation with the consulting surgeon of the theater, the army surgeon and his surgical consultant, commanding officers of hospitals, and the chief of the surgical service is fundamental to the successful operation of the group. The control of this highly specialized surgical skill and talent must be meticulous, and rest periods must be provided for the personnel permitting freedom from arduous field duties. Conservation of this personnel is urgently required as there are few qualified replacements available. The procedure which has been found most practical in the detail administration of the employment of the teams is as follows: Request for teams is made to the theater surgeon by an army or task force and these teams are furnished by the group. When attached to an army the request for teams is made to the army surgeon by the hospital commander. These requests are coordinated by the army consulting surgeon and head of the surgical group to ascertain where the available teams may be most efficiently employed. Requests from base section surgeons are made to the theater surgeon when teams are needed in base section hospitals and coordination is carried out as described. The teams are placed on temporary duty with the requesting hospitals and are relieved from duty when their mission has been accomplished or when they are more urgently needed elsewhere.

SUMMARY

The use and control of thoracic surgical teams of an auxiliary surgical group have been discussed. The requirements for members of the team are exacting and the need for general surgical training as the prerequisite for specialization in thoracic surgery is emphasized. The establishment of a small first priority surgical hospital adjacent to the division clearing station has furnished an excellent installation for the employment of surgical teams of an auxiliary surgical group in the management of nontransportable casualties. The thoracic surgeons have found a supreme challenge to their capabilities in these hospitals. They have also materially contributed to a better understanding and application of the fundamental principles of thoracic surgery by all surgeons engaged in forward surgery. The problems confronting evacuation hospitals in the handling of large numbers of thoracic wounded have been great and thoracic surgical teams have materially contributed to the solution of these problems. The use of thoracic surgical teams in fixed hospitals designated as thoracic surgical centers has permitted the completion of the reparative phase of surgery by the same surgeons that have dealt with the initial

phases of wound management of these patients in the forward hospitals. This has enabled the development of the broadest concepts relative to the management of war wounds of the chest. The thoracic surgical teams of an auxiliary surgical group have met the responsibilities in this specialized field of surgery in a most exemplary manner. It is gratifying to note the contrast between the present utilization of surgical teams of an auxiliary surgical group and the ideas of little over one year ago. The organization of a larger number of highly trained surgeons into surgical teams under one central control was new and untried. Few hospitals had considered the need for the additional assistance furnished by surgical teams; few, if any, had a plan for their employment and often the reception which the teams received was only cordial. Today, the usefulness of such an organization is fully recognized and appreciated by all who have had firsthand experience in the problems confronting the medical science of an active theater of operations.

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FULL-THICKNESS FLAP CLOSURE OF LARGE THORACOTOMY DUE TO CHEMICAL DESTRUCTION OF CHEST WALL

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IN THE presence of a large open thoracotomy resulting from trauma, an adequate respiratory exchange is maintained only with difficulty, which is usually increased by the presence of accompanying complications. Should the patient survive this major catastrophe, generally there will remain the problem of the empyema cavity, and in some cases, that of closure of the defect in the chest wall.

The case reported is one of destruction of a large portion of the chest wall by an injection of nitric acid in an attempt to commit suicide. There resulted a large defect in the left anterior thoracic wall which seriously affected the cardiorespiratory function. This was controlled by a large occlusive dressing with water-sealed drainage. The resulting empyema cavity was obliterated by a thoracoplasty. The defect over the precordium was closed by a full-thickness flap of chest wall. There resulted a satisfactory recovery. The case was complicated by other factors which are described in the case report.

CASE REPORT

A white man (Roper Hospital No. 92658), 23 years of age, was admitted May 10, 1937, at 2:05 P.M. He had been found in his room bleeding from the wrist and the neck. Also

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Received for publication March 27, 1945.

found were an empty vial which had contained phenobarbital, and a syringe and needle, and a partially empty bottle of nitric acid. Apparently he had taken a large overdose of phenobarbital, and had attempted to inject nitric acid into his heart. Upon admission he was drowsy and restless. There was an incised wound of the left neck exposing the thyroid gland, but missing the carotid sheath, and also a wound of the left wrist. These were cleansed and sutured. There were two needle puncture wounds in the left precordial region, and some soft tissue emphysema. He rapidly became comatose and remained so for several days, following which, apparently from the toxicity of the infection, he was irrational and excited with only occasional lucid intervals for a period of four weeks. For another three weeks he was at times irrational. The disturbed mental state severely taxed the low respiratory reserve. His temperature rapidly rose to 105° F. and remained elevated between 102 and 105° F. for one week, when it started gradually to subside. For the next eight weeks it ran a septic course reaching 100 to 101° F. daily. There was clinical and x-ray evidence of bilateral bronchial pneumonia after twenty-four hours. The soft tissue emphysema rapidly increased and within twenty-four hours involved the right neck and the left arm to the elbow. The presence of the soft tissue emphysema in the absence of a pneumothorax was best explained by an extrapleural injury of the lung. After twenty-four hours there was noticeable retraction of the precordium on inspiration and bulging on expiration. A precordial friction rub was noted. It was deducted that the acid had caused a subcutaneous necrosis of the chest wall. Within a few hours after admission the patient became cyanotic and his respiration became labored. There were frequent sinking spells. His condition remained precarious for several weeks. Oxygen was administered steadily for seventeen days, at first by nasal catheter and later by tent. For the next few weeks it was administered at frequent intervals because of labored breathing and cyanosis. The supportive and stimulative treatment consisted of fluids parenterally, coramine and caffeine. Prontosil in dosage of 10 c.c. every four hours was given for the first three days. Prontosil and prontylin were used later also in the course of the illness. He was given twenty-two blood transfusions varying

Fig. 1.—About May 25, 1937, slough of soft tissues, as yet incomplete; ribs left for support of dressing. Wound side of dressing shown at upper left.

from 250 to 500 c.c. each. On May 20, his condition rapidly became worse, at which time fluid was detected at the left base and 700 c.c. of slightly cloudy serosanguineous fluid were withdrawn. Streptococci and pneumococci were cultured. The following two days 660 and 420 c.c. were aspirated. On May 22, to provide more adequate drainage, the thinner precordial tissue was incised with resultant severe respiratory difficulty. A firm occlusive dressing of truck tire inner tube reinforced with sponge rubber was applied and through it a catheter was passed for water-sealed drainage. The drainage continued profuse and became purulent. Azochloramid irrigations were effective in reducing its volume and purulent character. For the next few weeks the tissues of the precordial region rapidly became gangrenous, increasing the size of the thoracotomy. In spite of his being in an oxygen tent, removal of the dressing for care of the wound resulted in extremely labored breathing and cyanosis. May 29, 1937, an intercostal closed thoracotomy was established in the seventh interspace postaxillary line, to provide more direct drainage. Gradually the wound edges became clean and the empyema cavity established so that oxygen therapy could be dispensed with. On June 12, a metastatic abscess on the right lateral thigh was incised for drainage. Until June 18, the patient steadily became weaker and it looked as though recovery were impossible. On August 13, he was discharged from the hospital with an open thoracotomy 10 by 10 cm. in size, situated to the left of the sternum and extending from the third to the seventh rib inclusive, and an empyema cavity occupying one-third of the left hemithorax. He was in an excellent frame of mind. The irrigations of the cavity with azochloramid and the occlusive dressing were to be continued.

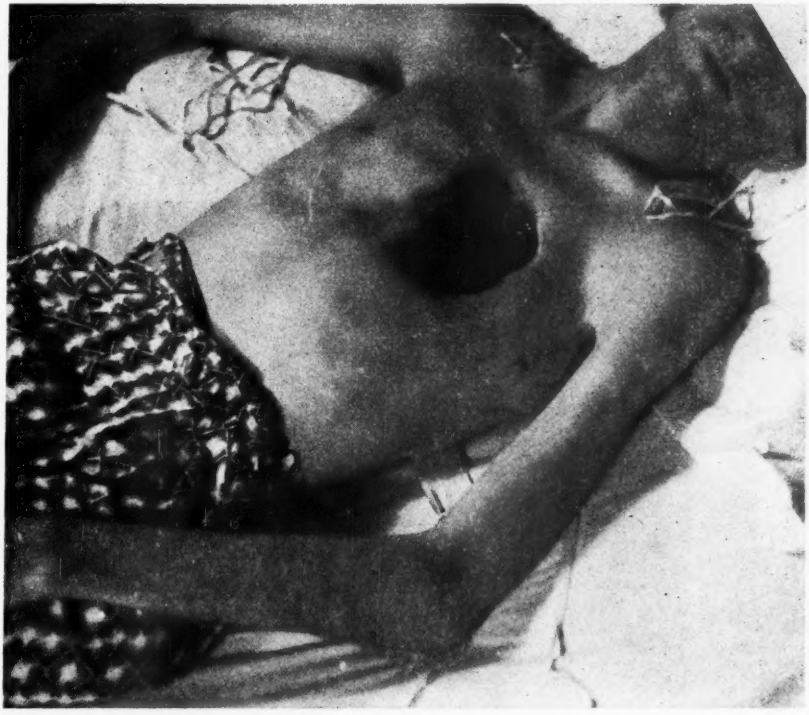

Fig. 2.—October, 1937, thoracotomy exposing pericardium and empyema cavity.

He was readmitted Oct. 23, 1937, at which time his general condition had greatly improved. The anterior thoracotomy was of the same size, however the empyema cavity was much smaller, the lung having expanded at the apex and the base. Oct. 28, 1937, a paravertebral thoracoplasty was performed consisting of the subperiosteal resection of about 10 cm. of the fourth to ninth ribs, inclusive. This practically obliterated the empyema cavity. Nov. 16, 1937, an operation was performed for closure of the defect over the precordium by making an intercostal incision through the full thickness of the chest wall lateral to the thoracotomy, between the third and fourth ribs, and a similar incision between the seventh

and eighth ribs. The mobility resulting from the recent paravertebral subperiosteal resection permitted sliding the full-thickness flap over the precordium, and holding it in place by means of sutures to the sternum and a circular chest bandage. He was discharged Dec. 5, 1937, with the empyema cavity almost obliterated and the precordium covered by a full thickness of chest wall. There remained a small sinus tract to the pleural cavity.

March 16, 1941, he was enjoying good health and engaged in active but not strenuous practice of his profession. The chest wall was firm except for a small area above the precordium which was thin and bulged slightly on straining. Several centimeters to the left of

Fig. 3.—March, 1941, full-thickness flap closure of defect in chest wall.

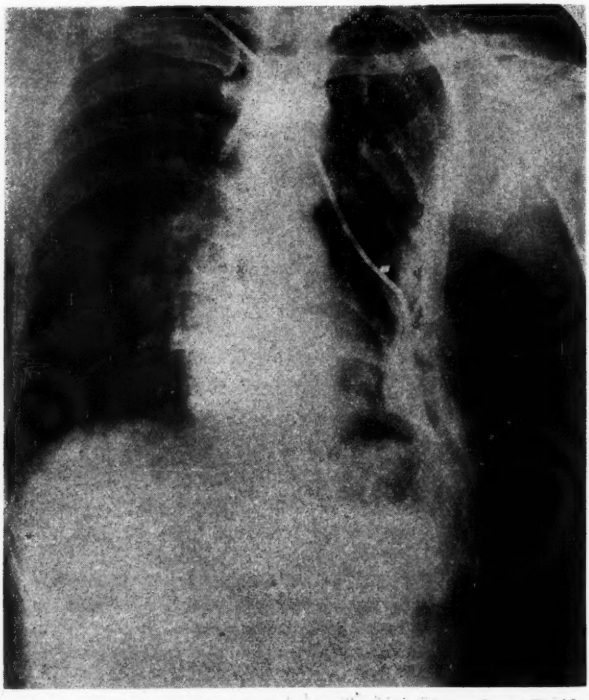

Fig. 4.—March, 1941, catheter entering sinus tract at site of marker. Residual pocket injected with x-ray opaque medium.

the sternum there was a small sinus tract, shown by x-ray examination following injection to be situated between the tenth and eleventh ribs in the midaxillary line. It was advised that this cavity be drained by an overlying thoracotomy.

A letter received March 22, 1945, revealed his condition to be essentially the same. He was working hard and hopes soon to be able to have the residual cavity obliterated.

SUMMARY

A case is reported in which a thoracotomy 10 by 10 cm. over the precordium, resulting from an injection of nitric acid, was closed by a combination of paravertebral thoracoplasty and a sliding full-thickness flap of chest wall.

PENICILLIN IN PULMONARY RESECTION

Preliminary Report

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IN RECENT years pulmonary resection has developed from a crude, chaotic procedure with a prohibitive mortality to a refined surgical operation with a mortality comparable to that of major abdominal procedures. Five factors have contributed to this rapid development, namely, (1) increased knowledge of thoracic physiology and pathology, (2) progress in anesthesia, (3) increased knowledge of shock, and fluid and blood replacement, (4) increased knowledge of surgical anatomy of the lungs, and (5) developments in chemotherapy.

The first factor has permitted the proper selection of patients for pulmonary resection, and has laid an intelligent foundation for their postoperative care. The second factor has abolished the fear of a surgically opened thorax through the employment of controlled positive pressure anesthesia, and has diminished the likelihood of bronchogenic dissemination of disease through the development of methods for tracheobronchial aspiration during anesthesia. Blood volume replacement, the third factor, has permitted the performance of prolonged operations involving great blood loss without the fear of shock. One of us (M. L. W.)[7] has determined the actual loss of blood in these operations and found, for instance, that the average loss of blood during a lobectomy is approximately 1,600 c.c. The fourth factor has made possible the development of the actual surgical technique of pulmonary resection. The anatomic studies of Blades and Kent,[1, 5] in which the vascular and bronchial systems of both lungs were described from a surgical viewpoint, popularized the individual ligation technique for the hilar structures in lobectomy in a manner similar to pneumonectomy.

The last factor, chemotherapy, has been the most recent development, coming after fairly general standardization of the technique of pulmonary resection. It has, of course, contributed to the control of infection, parenchymal, pleural, and chest wall. The sulfonamides have been used for at least five years, both preoperatively and postoperatively, locally in the pleural space as well as

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Received for publication June 1, 1945.

systemically. Postoperative pneumonia, one of the great hazards of pulmonary operations, has been adequately controlled, and pleural infections have been diminished by the use of the sulfonamides alone.

The discovery of penicillin has opened another new field in the control of postoperative pleural infections. White and his associates[5] have recently published indisputable proof of the value of penicillin employed systemically in the protection of the pleural cavity against infection. Reports from the war theaters by Nicholson and Stevenson,[6] and D'Abreu, Litchfield, and Scott[4] have shown the protective value of penicillin used both locally and systemically in conditions resulting from penetrating wounds of the chest, including sterile and infected hemothorax, empyema, and thoracotomy, especially for the removal of foreign bodies. D'Abreu and co-workers[4] in discussing war wounds of the chest stated, "a remarkable finding has been the capacity of the pleural cavity to retain instilled sodium penicillin for as long as three days; this means that a reservoir of the best known bacteriostatic agent can be maintained at the site of infection. Conversely, passage from the blood stream into the pleural cavity is slight. The method of intrapleural instillation is thus both more economical and far more effective than that of parenteral therapy, besides being less disturbing to the patient."

Blades[3] has recently expressed disappointment in the value of penicillin used locally and systemically in the protection of the pleural space against infection following pulmonary resection. His two series of cases, the penicillin-protected and the control series, had approximately the same incidence of empyema.

The purpose of this paper is to compare the course and result in patients with pulmonary resection treated with and without the administration of penicillin.

TECHNIQUE

Although this paper is not primarily concerned with the technique of pulmonary resection, a brief discussion of certain elements in the technique employed is indicated in order properly to interpret the results obtained.

An attempt was made to perform an individual ligation of the hilar structures in all cases. When this could be carried out, the vessels were isolated, ligated with heavy braided silk and secured with transfixion sutures of fine silk. The bronchus was closed with a single row of interrupted sutures of fine silk. An attempt was always made to cover the bronchial stump with the adjacent mediastinal pleura, and if such tissue was not accessible, a pedicled or free pleural flap was used.

Poorly developed fissures and scar tissue made the individual ligation technique impossible in some cases. In such cases a mass, or tourniquet, amputation technique was employed. The Shenstone tourniquet was used after making the hilar mass as small as possible by preliminary clamping and division of pulmonary tissue, as will be described. After amputation of the lobe, a transfixion suture of heavy silk was passed just distal to the tourniquet and the stump was thereby ligated in two segments. Supplementary hilar ligation was performed by isolating the large vessels and bronchi from the hilar mass as the amputation proceded, dissecting out and treating the vessels with ligatures of silk and the bronchi with interrupted silk sutures. The pulmonary tissues constituting the periphery of the hilar mass were then approximated to give a hilar stump partially or completely covered with visceral pleura. As with the individual ligation technique, bare areas in the stump were covered by adjacent mediastinal pleura.

Division of pulmonary tissue, either for segmental lobectomy or for the completion of poorly developed fissures, was accomplished between large curved hemostatic clamps. Fine chromic catgut on an atraumatic needle was used to form a line of simple continuous suture. Reinforcing mattress sutures of fine silk were used to reinforce and to pleuralize the line of incision.

Before the chest was closed, a stab wound not more than 1 cm. in length was made, usually in the ninth interspace in the posterior axillary line. Through this a No. 16 French catheter was inserted with its tip about 5 cm. within the pleural cavity. The open end of the catheter was clamped. The remaining lobe or lobes were then completely inflated with positive pressure at 10 cm. of water, and this inflation maintained as the chest was being closed. After the closure was completed, a 50 c.c. syringe with a three-way stopcock attached to the catheter was used to aspirate most of the remaining air in the pleural space. The aspiration was discontinued when a high negative pressure developed. The catheter was then clamped tightly with a screw clamp and incorporated in the dressing.

All lobectomies done prior to the use of penicillin were drained by rib resection and a large tube connected with a closed water seal system. This closed drainage was maintained for approximately two weeks, and then was converted to open drainage, usually with a smaller tube. In the patients treated with penicillin, drainage was not done.

In both series of cases whether or not penicillin was employed, the sulfonamides were used both locally and systemically. A single application of about 5 Gm. of sulfanilamide powder was used intrapleurally, with dusting of the drug around the hilar stump. Systemically, sulfadiazine in a dosage of 6 Gm. daily was employed for two or three days preoperatively and for a week postoperatively.

In the penicillin series, a solution of 100,000 units of sodium penicillin dissolved in 20 c.c. of sterile normal salt solution was used in both systemic and local administration. For two or three days before the planned operation 10,000 units of penicillin were given intramuscularly every three hours. This dosage was continued for three or four days postoperatively in the usual case, and for one week in cases in which the pleural cavity was heavily contaminated.

After the chest was closed and while the patient was still on the operating table, 10 c.c. of solution, representing 50,000 units of penicillin, were injected into the chest through the catheter. The catheter was left in place, clamped off, for forty-eight hours postoperatively to facilitate the aspiration of chest fluid, since during the first postoperative days the patient's discomfort makes thoracentesis difficult. Fifty thousand units of penicillin were daily injected intrapleurally for six or seven days after the operation. After the catheter was removed on the second postoperative day, these injections were made following daily thoracentesis, which was performed as long as there was any fluid present. Cultures were made from the fluid obtained at each aspiration.

MATERIAL

The material comprises forty-eight consecutive pulmonary resections carried out during a period of thirty-one months. In the first twenty-eight cases no use of local and systemic penicillin was made; in the last twenty cases penicillin was administered in the manner described. The two series were roughly similar in respect to diagnosis and operative procedure, except for differences in drainage as already described.

The twenty-eight cases prior to the use of penicillin constitute Group I, and the twenty cases in which penicillin treatment was used constitute Group II (Table I). There were twenty-five lobectomies and three pneumonectomies in Group I. Of the twenty-five lobectomies, fifteen were for bronchiectasis, four for tuberculosis, three for lung abscess, and three for cystic disease. Of the

TABLE I. MATERIAL

	GROUP I (WITHOUT PENICILLIN)		GROUP II (WITH PENICILLIN)	
	PNEUMONECTOMY	LOBECTOMY	PNEUMONECTOMY	LOBECTOMY
Bronchiectasis		15	3	11
Lung abscess		3		1
Tuberculosis		4	1	2
Cystic disease		3		1
Tumor	3		1	
Total	3	25	5	15

three pneumonectomies, two were for bronchogenic carcinoma and one for sarcoma (Table I). The pneumonectomies were all done by the individual ligation technique, and six of the twenty-five lobectomies in the same manner. Seventeen of the twenty-five required tourniquet amputation, one partial lobectomy had a clamp resection, and one case required a combination of individual ligation of the lower lobe and clamp resection of the lingula (Table II). Nineteen out of twenty-five, or 76 per cent, therefore, involved division of pulmonary tissue.

TABLE II. TREATMENT OF HILUS IN LOBECTOMIES

	GROUP I (WITHOUT PENICILLIN)	GROUP II (WITH PENICILLIN)
Individual ligation	6	3
Individual ligation with clamp division of pulmonary tissue	1	6
Tourniquet	17	5
Clamp	1	1
Total	25	15
Division of pulmonary tissue	19 (76%)	12 (80%)

There were fifteen lobectomies and five pneumonectomies in Group II (Table I). Of the five pneumonectomies, three were done for bronchiectasis, one for bronchogenic carcinoma, and one for tuberculosis. Of the fifteen lobectomies, eleven were done for bronchiectasis, two for tuberculosis, one (a partial lobectomy) for a pulmonary cyst, and one for lung abscess. The pneumonectomies were all done by the individual ligation technique.

Three of the fifteen patients with lobectomies had an individual ligation only, five had tourniquet amputation, three required a combination of individual ligation of the lower lobe and clamp resection of the lingula, three had individual ligation and clamp isolation of the hilum, and one with partial lobectomy had a clamp resection only (Table II). Therefore, twelve of the fifteen cases, or 80 per cent, had division of pulmonary tissue with grossly contaminated pleural spaces. This figure is in close approximation to the number of heavily contaminated cases (76 per cent) in Group I.

RESULTS

In Group I two patients with pneumonectomies had uncomplicated recoveries, while the third developed a bronchial fistula with a fatal empyema (Table III). In the same series, ten of the patients with lobectomies, or 40 per cent, developed bronchial fistulas postoperatively, and two required plastic

muscle flap closures after months of drainage. One pneumonectomy and all lobectomies developed empyema. The average time of tube drainage was twelve weeks.

On the other hand all patients with pneumonectomies in Group II had uncomplicated recoveries without bronchial fistula or empyema. Similarly, not a single patient with lobectomy in this series developed bronchial fistula and only one developed an empyema. This case will be presented in more detail later.

In the twenty-eight resections in Group I, there were four, or 14 per cent, subcutaneous wound infections (Table III). In Group II there were no chest wall infections.

TABLE III. POSTOPERATIVE COMPLICATIONS

	GROUP I (WITHOUT PENICILLIN)		GROUP II (WITH PENICILLIN)	
	PNEUMONECTOMIES (3)	LOBECTOMIES (25)	PNEUMONECTOMIES (5)	LOBECTOMIES (15)
Bronchial fistula	1* (33%)	10 (40%)	0	0
Empyema	1* (33%)	25 (100%)	0	1 (6.7%)
Chest wall infection	4 (14%)		0	

*These represent a single case with both complications.

The patients in Group II, exclusive of the three cases of tuberculosis, stayed in bed an average of eleven days postoperatively, and were hospitalized for an average of 22.9 days after operation (Table IV). These figures are in sharp contrast to the series not so protected in which the average postoperative duration of bed rest of all patients, except the four with tuberculosis, was 20.6 days, and the average postoperative hospitalization lasted 31.8 days. Most of the patients came from rural communities, sometimes from distances of several hundred miles, and could not easily get back to the hospital for attention to possible complications in the early weeks of postoperative convalescence. For this reason, it has been our policy to hospitalize these patients until the chance of complications is remote.

TABLE IV. MORBIDITY IN NONTUBERCULOUS CASES

	GROUP I (WITHOUT PENICILLIN)	GROUP II (WITH PENICILLIN)
Average postoperative day out of bed	20.6	11.0
Average postoperative day of discharge	31.8	22.9

The postoperative temperatures graphically shown in Fig. 1 represent an average of the daily recordings for the first fourteen days. The average febrile reaction in Group II is slightly but not strikingly less than in Group I.

In Group II aspiration of the chest on the first postoperative day resulted in an average recovery of from 400 to 500 c.c. of bloody fluid, and on the second day of from 300 to 400 c.c. From that time on the quantity decreased so rapidly that thoracentesis was not necessary as a rule after the seventh postoperative day. The color of the pleural fluid changed from bloody to serosanguineous to clear yellow. Cultures taken from every specimen of pleural fluid, except those from the single case of empyema, were negative. This was in marked contrast to the cases of Group I, in which empyema was common (100 per cent of lobectomies).

No significant comparison can be made between the mortality in the two series of cases. In the twenty-eight cases of Group I (twenty-five lobectomies and three pneumonectomies) there were three deaths. One patient with pneumonectomy for bronchogenic carcinoma had a poor bronchial closure due to a very high resection, developed a bronchial fistula and empyema, and died on the

twenty-fifth postoperative day. One patient with lobectomy performed for multiple lung abscesses died on the thirty-sixth day from suppurative pericarditis incidental to a small putrid basal empyema, and one with lobectomy for a basal tuberculous cavity died on the twentieth day from a fulminating tuberculous pneumonia in the contralateral lung. Penicillin might have prevented the deaths of the first two patients, but would not have influenced the outcome in the last case. In the twenty cases of Group II (fifteen lobectomies and five pneumonectomies) there was one death. One patient with lobectomy for a tuberculous cavity in the lower lobe died on the fortieth postoperative day from an extensive contralateral spread. Post-mortem examination revealed complete pleural healing on the side operated upon.

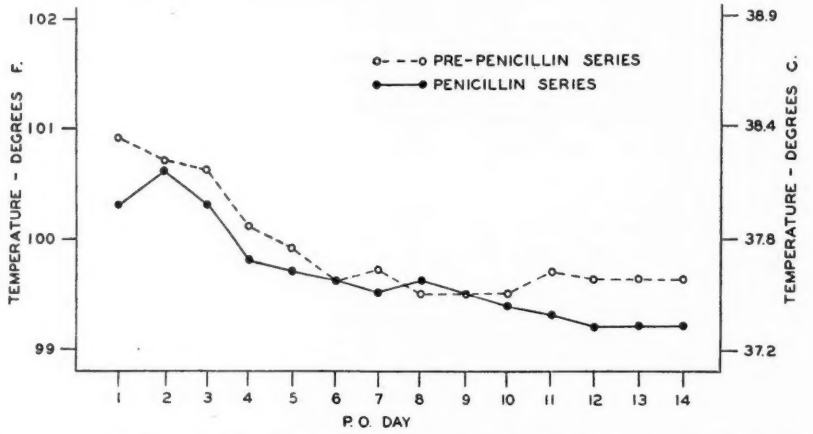

Fig. 1.—Showing the average daily temperatures for the first two postoperative weeks in the two groups.

The single case of empyema in Group II is worthy of special comment since it also represents a penicillin cure of empyema without open drainage. This patient, a 19-year-old girl, was admitted with the diagnosis of maxillary sinusitis and left lower lobe and lingular bronchiectasis of seven years' duration. A Caldwell-Luc operation was performed six weeks prior to a left lower lobe lobectomy and lingulectomy. At the time of the thoracic operation, dissection of the hilar structures was found impossible and a tourniquet was employed. Fifty thousand units of penicillin were given intrapleurally daily for the first postoperative week. Ten thousand units of penicillin were given intramuscularly every three hours for the first five days after the operation. Six pleural fluid cultures were negative but on the eighteenth postoperative day purulent fluid was aspirated. This culture showed *Streptococcus nonhemolyticus* and encapsulated gram-negative bacilli. For the next six days, 50,000 units of penicillin were injected into the pleura daily after the aspiration of small quantities of fluid. On the second day after the penicillin was begun the cultures became negative and the fluid much less purulent. The empyema cavity decreased in size until no more than 5 c.c. of clear fluid could be aspirated. Shortly after the operation the left upper lobe was found by physical and x-ray examinations to be completely re-expanded and at no time was the bronchus open. The patient was discharged in the sixth postoperative week in excellent condition. When she returned for a checkup three months after the operation, she was apparently completely cured.

It must be emphasized that the empyema cavity in this case was small, infection was in an acute stage, and the decreasing size of the cavity was followed by daily aspirations and x-ray studies. We agree with Blades, Hamilton, and Dugan[2] that penicillin does not alter the surgical treatment of empyemas which fail to obliterate rapidly with aspiration.

Another case of lobectomy warrants individual comment. This man, aged 45 years, was admitted with a large putrid lung abscess of three months' duration involving most of the right middle lobe. At operation the abscess was found to have extended across the transverse fissure into the upper lobe, and in dissecting the fissure, the cavity was inadvertently ruptured. One ounce of foul pus escaped into the pleural space. Resection of the middle lobe by tourniquet amputation and partial removal of the upper lobe by clamp division were carried out. In spite of extensive gross contamination no pleural or chest wall infection occurred.

DISCUSSION

In this paper we have attempted to show the advantage of using penicillin intrapleurally and intramuscularly in pulmonary resection without drainage of the pleural space. The evidence suggests that the intrapleural use of penicillin plays the most important role in the protection of the pleural space against infection. This is shown by the striking improvement in results when penicillin was added to and drainage subtracted from an otherwise fairly standard technique in a comparable series of cases. The improvement is indicated by a total absence of bronchial fistula as compared to an incidence of over one-third, by an almost total absence of empyema as compared to a high incidence, by the complete absence of chest wall infection as compared to an incidence of 14 per cent, and by an unmistakable shortening of bed rest and hospital stay. Only the mortality rate and the average postoperative febrile reaction are equivocal and both of these measurements slightly favor the penicillin over the pre-penicillin series.

The great difference between the results obtained by Blades[3] and the results in the present study must have some explanation. In his series of twenty-nine pulmonary resections, evelen patients, or 37.9 per cent, developed empyema, which was approximately the empyema rate of his control series, whereas in our series of twenty resections, one, or 5 per cent, developed empyema. We feel that the explanation lies in the fact that we do not drain the pleural space even for a few days, and can thus maintain a high concentration of penicillin therein by daily injections. The catheter which remains in the pleura for forty-eight hours is kept constantly clamped. Although we consider that the successful closure of the bronchus is important in the prevention of empyema, we do not agree with Blades that the incidence of empyema depends entirely on this factor. We would like to suggest the converse, namely, that empyema will contribute to the formation of a bronchial fistula. By maintaining pleural asepsis and maximum re-expansion of the remaining pulmonary tissue, particularly during the first postoperative week, the chances of bronchial fistula are lessened.

The catheter technique as we employ it in contradistinction to the usual closed drainage method seems worthy of re-emphasis. The clamped-off tube serves not for constant drainage but as a simple means of aspirating the fluid during the two days when the patient is most uncomfortable. The chief advantage of not draining the pleural space is that the intrapleural penicillin is allowed to remain in high concentration as long as desired. A second advantage

lies in the facilitation of re-expansion of the remaining pulmonary tissue by the negative intrapleural pressures induced and maintained by the aspiration of fluid immediately after operation and for several consecutive days thereafter. By favoring early re-expansion, the pitfalls of the unexpanded lobe are avoided. In all of the lobectomies in this series good expansion was obvious both by x-ray and by physical signs throughout the postoperative course. Should an empyema develop, it probably will be a late complication occurring at a time when the remaining lobe or lobes are completely re-expanded and are adherent to the chest wall. Under such conditions the empyema is apt to be small, and, if drainage is necessary, the patient's recovery should be rapid.

The striking demonstration of the value of penicillin lies in the heavily contaminated cases, that is, those in which lung tissue has had to be divided in the open pleura, the cases of clamp or tourniquet resection. It must be noted that in the present series 80 per cent of the cases in Group II had heavily contaminated pleurae. To this group can be added an additional case of bronchiectasis, the lobe being removed by tourniquet amputation by one of us (A. R. V.) while working in the Barnes Hospital, St. Louis, in 1944, a case which also presented no complications.

Finally, the patient's postoperative course is smoother with penicillin than without. He largely escapes an uncomfortable period of drainage. With shortening of his hospital stay his return to a normal life is speeded up. The mortality in a larger series can be expected to be consistently lower when the dangers of a putrid empyema with a bronchial fistula are decreased.

CONCLUSIONS

1. The use of penicillin in the pleura and systemically in association with a series of operations for resection of lung tissue without pleural drainage was followed by marked improvement in results in comparison with the results in a similar series without penicillin and with drainage.

2. Penicillin, locally and systemically, should play a part in the pre- and postoperative care of all patients with pulmonary resection.

3. When penicillin is employed in the pleura, postoperative drainage thereof is better omitted in order to maintain a high level of the drug locally and to favor re-expansion of the lung and bronchial healing.

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CYLINDROMA OF THE BRONCHUS

Report of Six Cases

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THE term "cylindroma" is not of recent origin. It was originated in 1856 by Billroth,[1] who first described an encapsulated orbital tumor that grossly had a glassy translucent appearance. Two types of cylinders could be demonstrated histologically, namely, the stroma which could be hyaline or myxomatous in character, and the parenchymal cells of the tumor which he regarded as being of epithelial origin. Von Ohlen,[2] in 1893, pointed out that the cylinders of epithelial cells actually enclosed cylinders of mucinous material.

A second form of cylindroma has been described by the dermatologists under the term "nevus epitheliomato cylindromatosus." These neoplasms are found only on the skin, most frequently the scalp, and are known as "turban tumors" (Ormsby and Montgomery).[3] Cutaneous cylindromas are regarded by many physicians as being closely allied if not similar to basal cell carcinomas. Indeed, Krompecher[4] suggested that cylindromas of mucous membrane surfaces and salivary and lacrimal glands are similar in appearance to basal cell carcinoma of the skin. This group of tumors appears to be different clinically and prognostically from cylindromas of mucous membranes and of salivary and lacrimal glands, such as were described by Billroth. Therefore, we are not considering cylindromas of the skin in this discussion.

According to Lemaitre,[5] cylindromas of the salivary and lacrimal glands and those in relation to the mucous membranes of the nose, with its paranasal sinuses, mouth, throat, larynx, and trachea have been described. Dockerty and Mayo[6] found that 18.5 per cent of primary tumors of the submaxillary gland were cylindromas. Stein and Geschickter[7] found that the incidence of cylindroma among primary tumors of the parotid glands was 17.4 per cent. Kramer and Som,[8] in 1939, stated that the gross features of cylindroma were (1) that the mucosa was intact over the tumor (if the tumor originated on a mucous membrane surface), (2) that there was a capsule around the tumor, and (3) that on cross section—the tumor presented a glassy gray appearance. They expressed the opinion that there was a marked tendency for local recurrence. This tendency for local recurrence and metastasis was evident in the series of tumors of the submaxillary gland reported by Dockerty and Mayo. In their cases there was a tendency for cylindromas to invade the lymphatic spaces around the nerves. On the other hand, Lemaitre stressed the absence of lymphatic involvement in this group of tumors. According to Beck and Guttman,[9] cylindromas are characterized by "slow growth, local progression, lack of metastases, prompt regression to irradiation and recurrence after local excision." Again, Lemaitre concluded that these tumors were not readily sensitive and therefore that treatment should be surgical.

Received for publication July 26, 1945.
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Enclosed in the cylinders of cells is a secretion which, according to Kramer and Som, is either mucus or hyaline material. Some observers have reported that this material does not stain with mucicarmine and others have reported that it is mucicarmine-positive.[5]

The first reported case of cylindroma of the lung was that of Heschl[10] in 1877. Kramer and Som reported three cases of cylindroma of the lung. In one of these cases the condition was not recognized as cylindroma until after

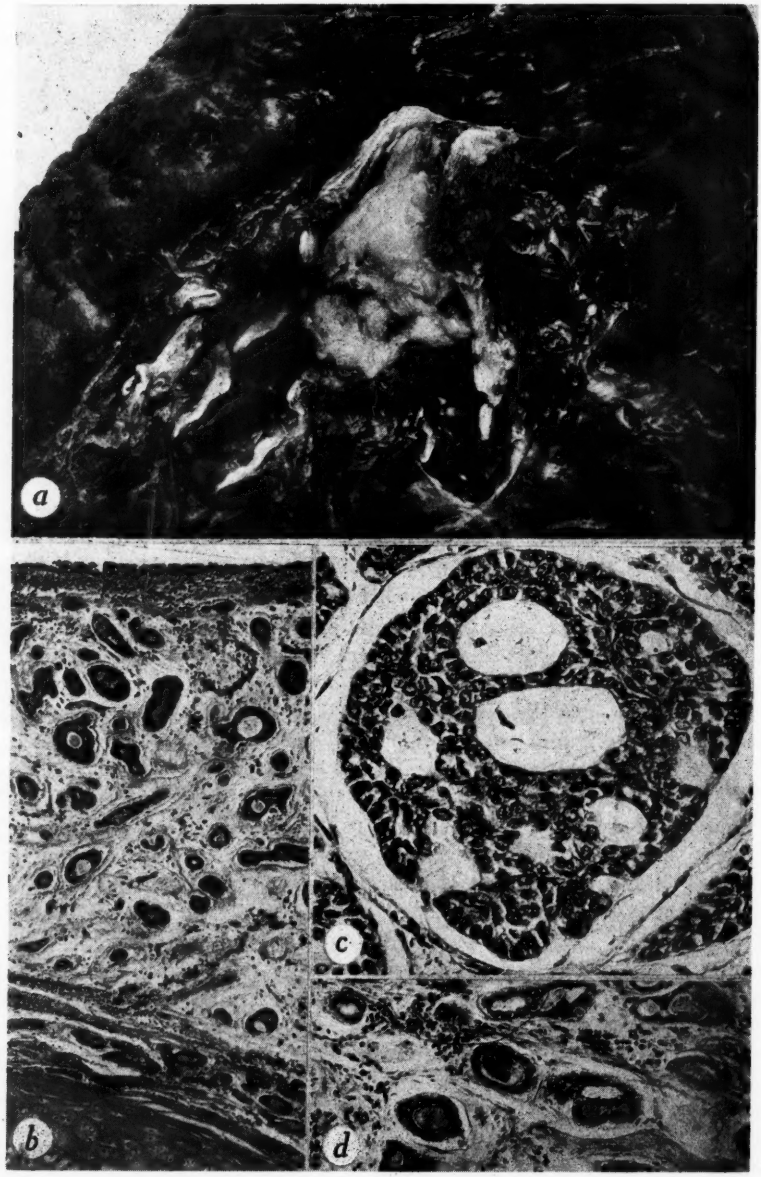

Fig. 1 (Case 1).—*a*, Cylindroma of the lung. The polypoid tumor, which projected into the left main bronchus, showed *b*, intact surface epithelium and tubular formation (×65), *c*, cylindroid or Swiss-cheese pattern (×265), and *d*, that the secretion within the tubules is mucicarmine-positive (×100). Note the mucoid connective tissue around the epithelial islands.

death of the patient from hemorrhage. The other two patients had had no recurrence two and three years, respectively, after bronchoscopic therapy. Kramer and Som noted that the gross appearance of the cylindroma of the lung in their cases was comparable with that of adenoma of the lung. Jacob, Delarue, and Gaultier,[11] in 1939, reported a case of cylindroma of the bronchus in which marked regression of the tumor was obtained after interstitial radium therapy. Jacob, Delarue, Huet, and Depierre,[12] in 1941, reported that this patient had died. At necropsy evidence of bronchiectasis but no metastasis was apparent. Madelung,[13] in 1941, reported two cases of cylindroma of the lung. In one of his cases the tumor was removed bronchoscopically and the patient was well two years later. The other patient died before treatment could be instituted and necropsy revealed no metastasis but extensive bronchiectasis and infection in the lung.

REPORT OF CASES

CASE 1.—A man, 36 years of age, was admitted to the Mayo Clinic in February, 1942. One year previously dyspnea had developed but he did not become incapacitated until one month before admission, when he had a violent paroxysm of coughing and expectorated a large amount of blood. Hemoptysis was so severe that three transfusions were necessary. This was followed by two more attacks of cough and hemoptysis.

Examination of the thorax at the time of admission revealed decreased expansion of the left side of the thorax and diminished breath sounds over the left lower thorax posteriorly. The heart was displaced to the left. A roentgenogram of the thorax showed complete flat density over the left side and displacement of the heart and mediastinum to the left. Bronchoscopic examination revealed that a large tumor was obstructing the left main bronchus just below the bifurcation of the left upper lobe bronchus.

Left pneumonectomy was performed and the patient had an uneventful convalescence. On examination in September, 1942, he was asymptomatic.

The pathologists reported that examination of the left lung showed a polypoid tumor mass projecting into the main bronchus with definite peribronchial extension (Fig. 1*a*). The tumor mass measured 2.5 by 2 by 1.5 cm. The entire lung showed marked bronchiectasis with chronic pneumonitis. No lymph nodes were involved. Histologic examination revealed that the tumor mass was a typical cylindroma. The cells were all small, regular, and round with few mitoses in evidence. The cells grouped themselves into two forms, namely, tubules (Fig. 1*b*) and the typical cylindroid or Swiss-cheese pattern. In the latter, there were punched-out holes which resembled Swiss cheese (Fig. 1*c*). In some parts this material showed an affinity for mucicarmine (Fig. 1*d*); in other parts it did not. The contents of the lumina stained pinkish or had a slightly basophilic tinge in hematoxylin-eosin preparations. Surrounding the epithelial cells there was a small amount of myxomatous connective tissue stroma such as is seen in mixed tumors.

CASE 2.—A man, 65 years of age, came to the clinic in November, 1936, because of cough and dyspnea. He was well until September, 1932, when an attack of chills and fever developed. These episodes of chills and fever had recurred frequently. Three years before admission he began to have cough and dyspnea which became progressively worse. Dyspnea frequently was associated with wheeze. Cough became productive of a large amount of purulent sputum.

On physical examination there was dullness to percussion over the right portion of the thorax and the breath sounds were diminished. The heart was displaced to the right. Roentgenogram of the thorax showed marked displacement of the heart and mediastinum to the right with almost complete opacity of the lower three-quarters of the right lung field. Bronchoscopic examination revealed obstruction of the right main bronchus. There was so much purulent secretion that it was impossible to visualize the lesion. Five hundred cubic centimeters of extremely foul, purulent material were aspirated.

After bronchoscopic aspiration there was some symptomatic improvement for several days, but the patient suddenly became weak and died on Dec. 5, 1936.

At necropsy a polypoid tumor which measured 3.5 by 3 by 3 cm. was found in the right main bronchus. It had extended beyond the bronchial wall and caused obstruction of

all the branches of the bronchus to this lung. There was severe bronchiectasis with abscess formation. The pleura was markedly thickened (Fig. 2*a*). There was no regional lymph node involvement nor metastasis elsewhere in the body. Histologic examination revealed round, regular tumor cells. Mitosis was difficult to demonstrate. The cells were making a faintly basophilic substance which in some regions was mucicarmine-positive (Fig. 2*b*). In no portion was a cylindroid or Swiss-cheese pattern apparent. This neoplasm represented an atypical cylindroma. There was hyalinization of the stroma around the epithelial cells.

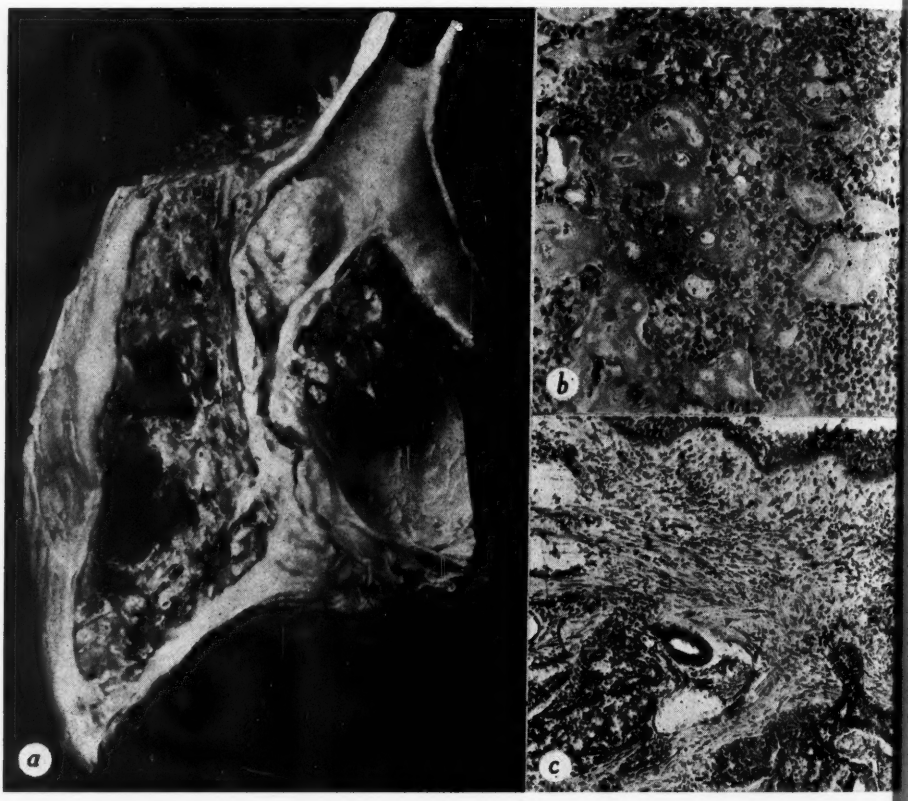

Fig. 2.—Cylindroma of the right main bronchus. *a* (Case 2), The polypoid characteristics of the tumor, bronchiectasis with abscess formation distal to the obstruction and thickening of the pleura are shown; *b* (Case 2), the small regular cells and the mucoid secretion can be seen; *c* (Case 3), cylindroma of the bronchus showing the tubular formation and the intact surface epithelium.

CASE 3.—A man, 51 years of age, came to the clinic in January, 1941, because of cough and hemoptysis. He had been well until February, 1938, when chills and fever developed. Temperature was 104° F. This acute illness was diagnosed pneumonia. After this he had wheeze and occasional hemoptysis. In June, 1938, he again had fever, and a cough which was productive of purulent sputum; from then to June, 1940, he had six such episodes. In August, 1940, the wheeze became progressively worse until he had intractable asthma.

On physical examination there was stridor and marked decrease in expansion of the thorax bilaterally. On bronchoscopic examination the patient stopped breathing when the bronchoscope was introduced. The trachea was found to be completely obstructed by a huge tumor mass which also involved the right and left main bronchi. A large portion of the tumor was removed and artificial respiration was instituted. Respiration soon returned to normal.

Specimens from the trachea and the right and left bronchi showed an identical morphologic appearance. The tumor cells were quite regular and round and showed few mitoses. Both the tubular and the cylindroid types of pattern were seen. The epithelium overlying the tumor was intact (Fig. 2*c*). The secretion within the lumen of the tubules, as well as

the cylindroid or Swiss-cheese regions, was slightly positive for mucicarmine. The stroma around the epithelial islands was hyalinized.

CASE 4.—A woman, 42 years of age, came to the clinic in April, 1927. Since she was 8 years of age she had complained of cough, dyspnea, and wheezing.

Examination of the thorax revealed coarse, musical râles throughout. Roentgenogram of the thorax showed a circumscribed nodule in the upper mediastinum on the right side. On bronchoscopic examination, the right main bronchus was found to be almost completely obstructed by a necrotic bleeding mass. Pus was exuding from the bronchus. The routine pathologic diagnosis on the tissue that was removed from this mass was adenocarcinoma, grade 1 (Broders' method), with evidence of tubular formation. Roentgen therapy was given over the thorax and for ten weeks the cough and wheezing disappeared.

She returned to the clinic in July, 1928. Cough, dyspnea, and wheezing had become progressively worse. She also had noticed some bloodstreaked sputum and had had several attacks of chills and fever. Roentgenogram of the thorax showed infiltration of the middle third of the right lung. The heart was displaced to the right side. Roentgen therapy again was administered. The patient died in 1929. Necropsy was not performed.

Review of the histologic sections revealed that the tumor was a typical cylindroma. The epithelium overlying the tumor was intact and ciliated in type. The neoplastic cells, which were small and regular in shape, tended to enclose a small lumen containing a slightly basophilic staining substance (Fig. 3*a*). Because of the small amount of tissue it was not possible to determine the affinity of this substance for mucicarmine. Mitotic figures were not apparent.

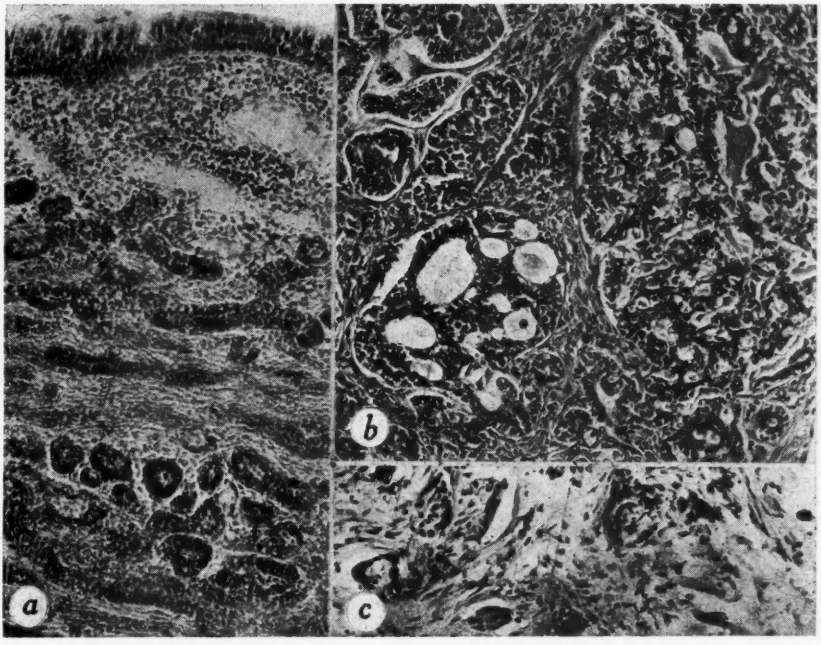

Fig. 3.—*a* (Case 4), cylindroma of the bronchus, showing tubular formation and intact surface epithelium (×125); *b* (Case 5), cylindroma of the bronchus, showing the cylindroid or Swiss-cheese pattern; the secretion in this pattern is of a hyaline nature (×115); *c* (Case 5), cylindroma of the bronchus, showing mucoid connective tissue around the epithelial islands (×115).

CASE 5.—A man 31 years of age, came to the clinic in December, 1943, because of cough and recurring attacks of chills and fever. He had been well until September, 1942, when cough, dyspnea, and wheezing developed. During the next five months, he had several attacks of chills and fever. In February, 1943, pain in the left thorax developed. He suddenly expectorated 100 c.c. of blood and the sputum had been bloodstreaked ever since.

On examination of the thorax, breath sounds were absent on the left side. Roentgenogram of the thorax showed massive atelectasis on the left. On bronchoscopic examination

a large tumor mass was found completely obstructing the left main bronchus at the junction with the trachea. About one-half of the tumor was removed with forceps and the base of the tumor was destroyed by means of surgical diathermy. Routine microscopic examination of the tissue removed bronchoscopically showed it to be a cylindroma.

One week later another bronchoscopic examination was performed. There was still a large tumor in the left main bronchus. This was removed with forceps and by means of surgical diathermy. The patient was given a course of roentgen therapy over the thorax.

When he returned to the clinic in July, 1944, bronchoscopic examination revealed that the lesion had almost completely disappeared, although the dyspnea was more marked.

He died in September, 1944. Necropsy was not performed.

This tumor histologically presented the appearance of a typical cylindroma. The epithelial cells forming the tumor were small and regular, practically devoid of mitoses. The tumor cells arranged themselves into two general patterns. The majority of the tumor cells were forming small tubules lined oftentimes by a single layer of cells in the center of which was a small lumen. The lumen of these tubules was filled with either hyaline material or material which stained slightly basophilic with hematoxylin. The alternate grouping of the cells is more characteristic of cylindroma. Here, they arranged themselves into cylinders with multiple cleanly punched-out holes in the cylinder of cells which contained a material which was faintly basophilic. This cylindroid pattern is suggestive of the appearance of Swiss cheese (Fig. 3b). The secretion in the lumen of the tubular formation showed an affinity for mucicarmine; that in the cylindroid or Swiss-cheese pattern showed a minimal or negative affinity for mucicarmine.

CASE 6.—A man, 48 years of age, was admitted to the clinic in November, 1937. For seven months he had noticed pain in the left side of the thorax, dyspnea, and hemoptysis which had started acutely with temperature of 104° F. Roentgenogram of the thorax showed diffuse infiltration in the left lower lobe. On bronchoscopic examination a tumor mass was found to be partially occluding the left lower lobe bronchus. On microscopic examination of the tissue, the report was adenocarcinoma, grade 1, of the mucous glands, comparable to an adenoma. It was possible to remove most of the tumor with forceps. The base of the growth was destroyed by means of surgical diathermy.

The patient returned to the clinic in April, 1938, at which time his general health was improved. Cough had persisted and he was expectorating thick green sputum. Roentgenogram of the thorax showed little change from the one made in November, 1937. Bronchoscopic examination showed that the tumor had increased in size and was almost completely obstructing the left upper lobe bronchus. Radon needles were inserted into the base of the tumor (five 1 millicurie radon seeds were implanted).

He returned to the clinic in August, 1938, at which time bronchoscopic examination showed that the tumor had decreased about 50 per cent in size. It was removed and the base was destroyed by means of surgical diathermy.

The patient returned to the clinic in November, 1943. Aside from an occasional cough he had been asymptomatic since his previous visit. On bronchoscopic examination, a small recurrence was found in the left upper lobe bronchus. This was destroyed by means of surgical diathermy.

Re-examination of the tissue removed bronchoscopically revealed an atypical cylindroma. The cells were small and irregular and were producing a slight basophilic secretion suggestive of mucus. Mitotic figures could not be demonstrated. In between the epithelial cells was a generous amount of myxomatous stroma which had the characteristics of the precartilaginous stroma seen in mixed tumors (Fig. 3c). The epithelium overlying this neoplasm was intact. The neoplasm appeared to be arising from the mucous glands in the bronchial wall.

COMMENT

Pathology.—In our series of bronchial neoplasms approximately 10 per cent belonged to the adenoma and cylindroma groups. Forty-four adenomas or cylindromas of the bronchus have been encountered. Thirty-eight of these proved to be adenomas. The six cylindromas which are reported in this paper comprise 13.6 per cent of the combined adenomas and cylindromas. The adenomas have previously been reported.[14]

On superficial examination it would appear that cylindroma of the bronchus differs from adenoma of the bronchus mainly on histologic examination. The

gross appearance and course of the diseases are similar; however, clinically there are several suggestive differences. One of these is the tendency of cylindroma of the bronchus to involve the trachea, a feature which was encountered in case 3. Adenomas of the bronchus, on the other hand, rarely involve this organ. This is not surprising since cylindromas comprise 30 per cent of tumors of the trachea as reported by us;[15] on the other hand, adenomas were not encountered in this group of neoplasms. It is apparent from Case 1 that cylindromas of the bronchus can produce obstruction of the bronchial tree with resultant bronchiectasis and abscess formation which can result in death. Such a case in which death resulted from bronchiectasis was reported by Jacob, Delarue, Huet, and Depierre. In one of the cases reported by Kramer and Som, death was due to pulmonary hemorrhage. It is our impression that cylindromas are somewhat more infiltrative than adenomas. However, in none of our cases nor in those reported in the literature was there any evidence of metastasis. It is noteworthy that most observers express the opinion that cylindromas are sensitive to irradiation therapy.

As has been stated, the gross appearance is similar to that of adenoma. The tumor, which almost invariably arises from a large rather than a small bronchus, forms a polyp projecting into the bronchial tree. In the two cases in which the lung was available for study the extrabronchial portion of the neoplasm was quite sizable. As has been stated, one of these was removed surgically, the other was evident at necropsy. This is also true in adenoma of the bronchus. The tumor was sharply demarcated from the surrounding uninvolved bronchus and lung.

The cylindroma histologically is similar to the adenoma in that the cells comprising the neoplasm are small and regular but often hyperchromatic. Mitoses in both types of tumors are difficult to find. The mucous membrane overlying the cylindroma is almost always intact. The main point of differentiation from adenoma is a secretion apparently being produced by the epithelial cells. In our experience, this secretion, which must be some kind of mucus, sometimes has stained with mucicarmine. Adenoma of the bronchus does not produce a mucoid secretion.

The myxomatous or precartilaginous connective tissue in some of these tumors is consistent with that encountered in cylindromas in the salivary glands and in relationship to mucous membrane surfaces of the nose, throat, mouth, and trachea. This peculiar connective tissue suggests a relationship to mixed tumors. We never have seen a true mixed tumor of the bronchus, although Womack and Graham[16] suggested that adenomas of the bronchus were closely allied with mixed tumors. Two types of histologic appearance have predominated in cylindromas in our group of cases: (1) the tubular formation in which a single or double layer of cells lines a small lumen which is filled with mucinous secretion and (2) the cylindroid pattern in which there are cylinders of cells as well as mucinous or hyaline secretion. This appearance is suggestive of Swiss cheese.

The origin of these neoplasms in the bronchus is not clear. However, in Case 6 the neoplasm appeared to originate from the mucous glands in the wall of the bronchus.

Symptoms.—Unfortunately, tumors of the bronchus do not give rise to any characteristic symptoms. In general the symptoms depend essentially on the situation of the tumor and the degree of bronchial obstruction it produces. The symptoms do not differ from those produced by any pulmonary inflammatory

disease. As in bronchiogenic carcinoma, the absence of characteristic symptoms is one of the greatest obstacles to the early diagnosis of cylindroma of the bronchus.

Cough is often the earliest symptom. At onset the cough is usually dry and nonproductive. As the tumor increases in size, it obstructs the lumen of the bronchus and interferes with normal drainage of the bronchi distal to the lesion. The retained secretions become infected by invading organisms, and bronchitis, bronchiectasis, or abscess may develop, depending on the character of the organisms. When secondary infection of these secretions occurs, the cough becomes productive and the sputum purulent.

When secretions become thick and tenacious or when marked edema of the bronchial mucosa is present, there may be so much plugging of the reduced bronchial lumen that the secretions are dammed back. The retained secretions are usually secondarily infected and give rise to attacks of chills and fever. Such episodes frequently are mistaken for attacks of pneumonia.

Hemoptysis may vary in severity from streaking of the sputum with blood to severe and almost fatal bleeding, as was encountered in Case 1.

Dyspnea occurs when the caliber of the bronchial lumen is so compromised that there is interference with the air entering and leaving the lung. This may occur as the result of intrabronchial enlargement or extrabronchial compression. Dyspnea associated with neoplasms of the bronchus often seems out of proportion to the size of the lesion and the degree of associated atelectasis. Dyspnea may occur suddenly but is usually gradual in onset. When it is associated with wheeze, an incorrect diagnosis of asthma often is made.

Although pain was noted in two of our patients, it was not the severe, sharp pain which so often is associated with bronchiogenic carcinoma. Pain was usually characterized by a sense of discomfort or pressure.

None of our patients had hoarseness.

Physical Findings.—The physical findings in cases of cylindroma of the bronchus, as in any neoplasm of the bronchus, are dependent on the size and situation of the tumor, the degree of bronchial obstruction, and the secondary inflammatory changes present. Since cylindromas usually arise in the larger bronchi, the findings elicited by physical examination are usually striking. Inspection of the thorax with the patient in the recumbent position is a valuable but frequently neglected procedure. Expansion of the chest on the involved side lags behind that on the unaffected side. Another valuable procedure is to listen carefully for a wheeze. This can be elicited by placing the examining ear close to the patient's mouth while the patient breathes with the mouth open. The patient should be placed in the recumbent, upright, and bending forward positions. One may elicit dullness on percussion over the involved region if the region of atelectasis is close to the thoracic wall. Suppression of breath sounds over the involved side is the most important finding on auscultation and usually indicates bronchostenosis. If secondary infection has developed in the retained secretions and bronchial obstruction is not complete, coarse râles may be associated with the suppression of breath sounds.

SUMMARY

Six cases of cylindroma of the lung have been reported. The clinical course was remarkably similar to that of adenoma of the bronchus with the exception that in one case the neoplasm involved the trachea. The bronchoscopic appearance of the tumor also was similar to adenoma of the lung. The

lung, which is the seat of a cylindroma, grossly resembles that in which an adenoma is found in that bronchiectasis and lung abscess are common sequelae to the long-standing obstruction produced by the tumor. Cylindroma histologically presents a different appearance from adenoma of the bronchus and appears to be more closely allied to mixed tumors.

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SPONTANEOUS PNEUMOTHORACES OCCURRING IN PATIENTS UNDERGOING PERORAL ENDOSCOPY

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THE common complications of bronchoscopy are well known. Jackson[1] has called attention to the danger of direct injury to the bronchus and underlying vessels. Myerson[2] described a case of fatal injury at bronchoscopy in which the bronchus was perforated and the underlying pulmonary artery torn. The dangers attendant to anesthesia, both local and general, have been frequently described. The most common cause for postoperative alarm is the occurrence of subglottic edema;[1-3] this also is well known. Little if any attention has been drawn to the association of pneumothorax as a bronchoscopic hazard.

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Received for publication Aug. 15, 1945.

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Two cases of direct trauma to the bronchial tree with resulting collapse of the lung and subcutaneous emphysema were mentioned by Kahler.[3] Both these instances were in attempts at the removal of foreign bodies. Soulas[4] described emphysema of the neck and thorax occurring in conjunction with the removal of a foreign body in the right main stem bronchus. A similar instance was described by Benjamins.[5] The mechanism of leakage of air into the mediastinum in cases of sharp-pointed foreign bodies is evident.

Our interest in this subject has been aroused by the occurrence of pneumothorax following diagnostic peroral endoscopic examination. Furthermore, none of the five examples we describe in this communication were associated with the use of biopsy forceps. A careful study of the literature has revealed only one case which might be similar to our own. However, even this one may not be rightly so classified. In 1933 Carpenter[6] described a case of abscess of the right lower lobe in which "bronchoscopic drainage was done three times and pneumothorax developed on the same side and recovered promptly." No reference is made regarding the time relationship between occurrence of pneumothorax and the bronchoscopic aspiration. In 1935 Carroll[7] described two cases of pneumothorax following esophagoscopy. However, the fact that both of the patients developed pyopneumothorax rapidly suggests that some direct injury to the esophageal wall may have been the inciting factor.

The mechanism of the production of spontaneous pneumothorax deserves a brief review here. By far the most common etiology is tuberculosis.[8] The consensus of opinion attributes this to the rupture of a subpleural tuberculous focus. Other accepted causes include pneumonia, pulmonary gangrene, bronchiectasis, lung abscess, foreign body, empyema, tumor, congenital cysts, and pulmonary emphysema.

Laennec,[9] in his work published in 1819, considered the role of the emphysematous bleb and remarked, "It is likewise probable that in the case of the emphysema of the lungs, with rupture of some air cells and extravasation of air under the pleura, the membrane may sometimes be ruptured and the disease in question (spontaneous pneumothorax) be thus found."

The role of perforating injury to the chest wall or injury from within the bronchial tree by sharp objects or instruments in the production of pneumothorax needs no further comment.

One fully understands the possibility that pneumothorax following bronchoscopy should immediately make one conscious of the possibility that this complication may be secondary to direct injury to the visceral pleura by the bronchoscopist. However, we are also conscious of the fact that we are dealing with patients with diseased lungs in whom the stage is set for the occurrence of spontaneous pneumothorax even though no bronchoscopic examinations were done. That is to say, the bronchoscope is being introduced into the lungs in which subpleural suppurative foci or emphysematous blebs are present. Furthermore, patients who are bronchoscoped by thoracic surgeons are more likely to undergo periodic fluoroscopic examination which may reveal completely unsuspected pneumothoraces. This is further emphasized by the fact that the great majority of the cases will have sufficient adherence between the pleural surfaces to minimize symptoms of the complicating pneumothorax. This last statement is attested by the fact that two of the patients in the five cases reported herein had no respiratory difficulties to suggest the presence of air in the pleural

space. However, this happy state of affairs may not necessarily be present and, therefore, the complication may lead to dire results unless one is well conscious of it. It is for this reason that this communication is presented and the cases described in detail.

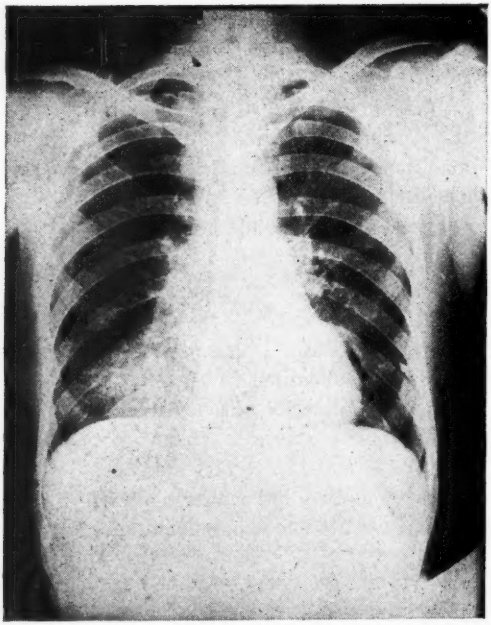
In regard to the frequency of this complication any figure may be misleading because fluoroscopy has not been made a routine follow-up of all bronchoscopic examinations. However, it might be mentioned that all four cases noted herein occurred within a twelve-month period during which time 537 bronchoscopic examinations were made on the surgical chest service for either diagnosis or therapy but not for the removal of foreign bodies.

It is important to emphasize that we feel that no major bronchi suffered direct injury in the cases herein described. This is attested to by the rapidity of healing and the lack of development of empyema in Cases 1, 2, and 6. The pyopneumothorax in Case 3 could be expected from spontaneous rupture of the peripheral lung abscess. We mean, therefore, to describe this occurrence of pneumothorax as a spontaneous phenomenon likely to appear in these types of cases, in which the patients are incidentally undergoing peroral endoscopy. Thus, we describe it not as a complication of the procedure but rather more as an indirect hazard of the coughing paroxysm subsequent to endoscopic manipulation.

CASE REPORTS

CASE 1 (B. H. F1043).—E. E., a 34-year-old white man, was admitted to the surgical service on Feb. 9, 1945, with the diagnosis of peptic ulceration of the lesser curvature of the stomach which was intractable to medical measures. A subtotal gastric resection was performed Feb. 24, 1945, and signs of atelectasis of the right lower lobe were noted the following day. Despite conservative measures and chemotherapy, the roentgenogram (Fig. 1) on March 12, 1945, revealed an abscess of the right lower lobe. Bronchoscopy was carried out on March 24, 1945, and revealed an inflammatory stricture of the anterolateral branch of the right lower lobe, plus an inspissated mucous plug. The latter was removed and about 30 c.c. of thick, foul smelling pus were aspirated from the abscessed cavity. The procedure was carried out under pontocaine anesthesia and during the next twelve hours the patient coughed excessively, producing considerable mucopurulent material. The next day the patient felt markedly improved and had suffered no dyspnea; the temperature fell from 39 to 37.4° C. However, physical examination suggested the presence of a pneumothorax, and fluoroscopic examination was carried out, showing 80 per cent collapse of the right lung due to pneumothorax. An x-ray view (Fig. 2) corroborated this finding. The patient developed no fluid and with continued chemotherapy showed dramatic resolution of the disease. The interpretation was made that the pneumothorax was secondary to rupture of an emphysematous bleb, resulting from the severe postbronchoscopic coughing paroxysms. For this reason no further aspirations were carried out as long as the patient's course was satisfactory; he was discharged cured three weeks later and subsequent follow-up has failed to reveal any evidence of recurrence.

CASE 2 (St. Louis Children's Hospital B-876).—J. F. R., an 8-year-old white boy, was admitted to the hospital on May 28, 1945, with a history of chronic productive cough for four years, being underweight, and having frequent colds and night sweats. Physical examination revealed a complete atelectasis of the left lung. The bronchoscopic examination was performed on June 1, 1945, and a complete obstruction of the left main stem bronchus by edema and mucous plugs was found. The plugs were removed and excessive thick pus was obtained from both lobes. It was found necessary to do repeated bronchoscopic aspirations. The patient was fluoroscoped repeatedly to observe the return of aeration of the left lung. Lipiodol bronchograms were done, June 12, which showed minimal bronchiectasis of the lingula and left lower lobe with some persistent obstruction of two of the branches of the lower lobe. Gradual progressive improvement occurred. One June 24, two days following a bronchoscopic aspiration, fluoroscopy revealed the presence of an asymptomatic 50 per cent collapse of the left lung due to pneumothorax. There was no evidence of fluid



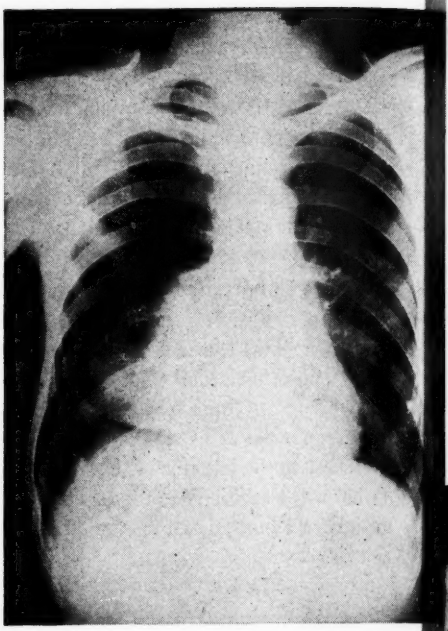


Fig. 1 (Case 1).—Roentgenogram of chest taken prior to bronchoscopy showing an abscess of the right lower lobe.

Fig. 2 (Case 1).—X-ray view of chest taken twenty-four hours following bronchoscopic aspiration. The adherence of the lower lobe to the diaphragm probably prevented dyspnea. Note the absence of fluid.

Fig. 3 (Case 2).—Lipiodol bronchogram which demonstrates small presistent pneumothorax occurring after repeated bronchoscopies. Fluid did not appear in this case.

in the pleural cavity, and four days later a bronchogram showed an essentially normal lung with about 10 per cent persistent pneumothorax (Fig. 3). The child has subsequently reached complete recovery. All bronchoscopies on this patient were done under avertin anesthesia.

CASE 3 (No. 119202).—J. C. McK., a 42-year-old white man, was admitted to the Barnes Hospital on Nov. 2, 1944. History revealed the appearance of a lung abscess in the right upper lobe eighteen days following an hernioplasty performed under general anesthesia, Aug. 6, 1944, at another hospital. Physical examination revealed a chronically ill man with clubbing of the fingers and signs of suppuration in the right upper lobe. The x-ray film (Fig. 4) revealed the abscess to be situated in the paravertebral area of the right upper lobe. On Nov. 3, 1945, the patient was bronchoscoped under avertin anesthesia and considerable pus was found to be coming from the right upper lobe orifice. No evidence of tumor was noted. The patient returned from the operating room quite cyanotic and with labored breathing. He complained of severe pain in the right side of the chest and examination revealed signs of fluid and air in the right pleural cavity. Despite penicillin therapy a frank pyopneumothorax developed, which was finally treated by open thoracotomy drainage on Nov. 13, 1945. A roentgenogram (Fig. 5) taken on November 10 showed evidence of a fluid level on the right side. The patient gradually improved and after a revision of the thoracotomy drainage on Dec. 28, 1944, recovery was complete from the lung abscess and empyema.

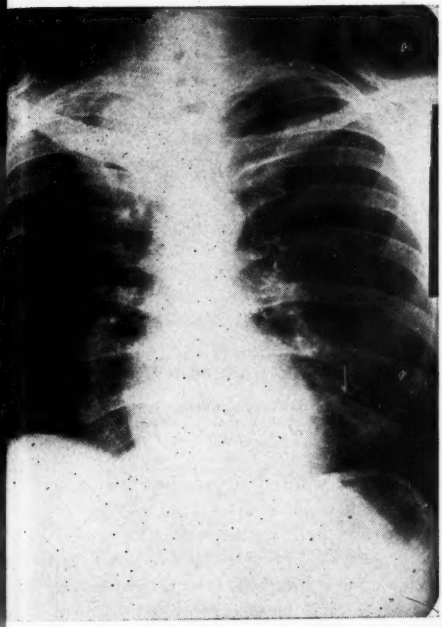


Fig. 4 (Case 3).—This film demonstrating opacity in right upper lung field was taken prior to bronchoscopic aspiration of the upper lobe abscess.

Fig. 5 (Case 3).—Roentgenogram of chest showing the presence of a right pyopneumothorax occurring after bronchoscopic aspiration.

CASE 4 (F5425).—E. G. H., a 29-year-old white woman, was admitted to Barnes Hospital, July 19, 1945, with a history of known tuberculosis since 1932. She had pneumothorax and phrenemphraxis in the intervening years but the disease progressed to a complete stenosis of left main stem bronchus with secondary atelectasis of the left lung and marked emphysema of the right. The sputum remained strongly positive for tubercle bacilli. She was referred to us for possible left total pneumonectomy and a bronchogram (Fig. 6) was performed July 20. Bronchoscopic examination was performed under pontocaine anesthesia. The examination revealed severe distortion of the trachea with complete stenosis of the left main stem bronchus, while the right side appeared normal except for tracheal distortion. No immediate ill effects were noted but the patient coughed severely. About thirty minutes,

later the patient had a very severe episode of coughing followed by extreme dyspnea and progressive cyanosis. By the time we were able to get to her she was moribund. Examination revealed absence of breath sounds except for the upper anterior portion of the right lung. A combination of bronchoscopic aspiration and immediate closed thoracotomy tube drainage for a tremendous right tension pnuemothorax was done. Considerable mucus was found in the right main stem bronchus plus an immense amount of air in the right pleural space. However, the process proved to be irreversible and she succumbed rapidly. Unfortunately, post-mortem examination was not permitted.

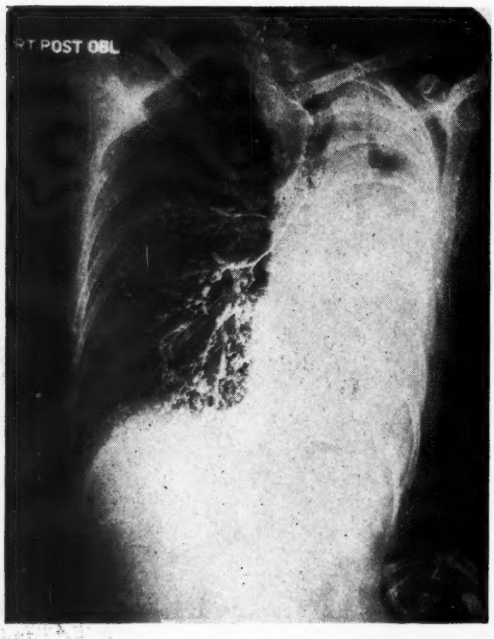


Fig. 6 (Case 4).—Lipiodol bronchogram demonstrating marked deviation of the trachea to the left and complete stenosis of the left main stem bronchus. There is marked emphysema of the right lung.

Fig. 7 (Case 5).—X-ray picture of chest showing a remarkable similarity to patient E. G. H. There is marked widening of the intercostal spaces on the right, tracheal deviation, and atelectasis of the entire left lung.

CASE 5 (B. H. 120210).—J. T. Z., a 35-year-old white woman, was admitted to Barnes Hospital on Dec. 10, 1944. Her history revealed known tuberculosis since 1936. Temporary arrest of the disease followed two years of sanatorium care. Eighteen months prior to admission she became subject to severe attacks of dyspnea and cyanosis in association with coughing paroxysms. She had been in the habit of keeping an oxygen tank at her bedside. She was sent to us for consideration of a possible left total pneumonectomy. The x-ray picture at the time of admission (Fig. 7) showed an extreme similarity to that in Case 4. Examination revealed a marked inspiratory and expiratory bronchial wheeze, with severe deviation of the trachea to the left, absence of breath sounds on the left, with signs of advanced emphysema on the right. Attempted thoracenteses failed to obtain fluid, which was suspected in the left pleural cavity. Bronchoscopic examination was carried out Dec. 12, 1944, revealing that "the trachea was very much displaced to the left. The carina was greatly distorted and the right main stem bronchus was thus partially stenosed due to distortion and could not be well visualized. The left main stem bronchus appeared to be almost completely stenosed." No ill effects were noted until six hours after bronchoscopy when sudden severe dyspnea occurred. Breath sounds were audible on the right but extremely shallow and wheezing. Despite nasal suction, oxygen tent, aminophyllin, the passage of an endotracheal tube, and the use of the Drinker respirator, the patient's condition became increasingly severe, with anoxemia, terminal convulsions, and death twenty-two hours following bronchoscopy. The possibility of tension pneumothorax was not suspected in this patient.

CASE 6 (No. 124332).—G. L. P., a 61-year-old white man, was admitted to the McMillian Hospital on May 18, 1945, because of symptoms of esophageal obstruction for two years. Barium x-ray studies revealed some dilatation of the esophagus with evidence of obstruction at the esophagocardiac junction. Esophagoscopic examination was carried out under pontocaine anesthesia by the otolaryngologic service and no intrinsic pathology of the esophagus was found except for evidence of cardiospasm. The patient was an unstable individual from the psychiatric angle and became uncontrollable as the scope was being removed. It was felt that injury of the esophagus or pharynx might have occurred during this episode. The patient rapidly developed severe dyspnea and subcutaneous emphysema of the neck tissues. The chest service was called into consultation and we found evidence of cyanosis, subcutaneous emphysema, and a complete left tension pneumothorax. Suspecting a probable rupture of the esophagus, an emergency exploratory thoracotomy was carried out by one of us (O. A. A.). Under positive pressure endotracheal anesthesia, the left pleural cavity was explored and the entire left lung was found to be collapsed. Examination of the mediastinal pleura revealed a 4 cm. longitudinal rent just below the arch of the aorta. No hemorrhage was present about this site. The entire thoracic esophagus was explored throughout its length and no visible injury to its wall found. A Levin tube had been placed into the esophagus prior to the operation and air was instilled through this into the esophagus with the mediastinum covered with normal saline solution. No air bubbles were noted. As a further test, methylene blue was instilled through the Levin tube and no evidence of it was visible in the left pleural cavity. The mediastinal pleura was left wide open and the chest closed with catheter drainage under water seal. The superior mediastinum was explored bilaterally through the neck and drained with Penrose drains. Subcutaneous emphysema was encountered but no evidence of extravasation of the instilled methylene blue could be seen. Prophylatic penicillin was given, and the postoperative course was uneventful and afebrile. All drains were removed by the eighth postoperative day. In view of the lack of postoperative complication and the absence of any direct evidence of esophageal perforation, it is postulated that this patient may have developed mediastinal emphysema with rupture of mediastinal pleura and left pneumothorax secondary to perforation of the pyriform sinus or even spontaneously. This patient was discharged on June 6, 1945.

DISCUSSION

The preceding six case histories consist of two cases wherein pneumothoraces occurring coincident to bronchoscopy were asymptomatic and these pneumothoraces were discovered accidentally by fluoroscopic examination. Both cases were in the presence of suppurative disease of the lung and no evidence of empyema occurred. The frequent association of areas of emphysema in relation to patchy atelectasis in children caused us to believe that the pneumothorax in the child (Case 2) was due to the rupture of an emphysematous bleb. We know from subsequent follow-up examination that one patient (Case 1) had some bilateral emphysema and we suspect a similar etiology here. The third patient had considerable suppurative disease on the surface of the lung at the time of bronchoscopy and probably a weakened necrotic area of the periphery of the abscess ruptured, producing secondary pyopneumothorax. The fourth patient had advanced tuberculosis and far-advanced pulmonary emphysema of the one functioning lung. Either or both of these etiologic agents may have played a role during her severe postbronchoscopy, coughing paroxysms.

Case 5 is not presented as an instance of postbronchoscopic pneumothorax but rather because of the extreme similarity of the x-ray films in this case (Case 4). We cannot be sure of the cause of death, but we feel it should be described as a complication of bronchoscopy and the coughing paroxysm which may accompany the procedure. A possible clue in regard to the difficulties may be found in the following quotation from Jackson:[1] "Compression stenosis of the trachea associated with pulmonary emphysema accounts for their dyspnea during the attacks of coughing."

The sixth case is described because of rarity and because it may carry out the premises of Hammen[10] put forth in 1937. He described seven cases of spontaneous mediastinal emphysema in which two advanced to the production of pneumothorax. Two possible modes of the production of the pneumothorax were described, namely: (1) The air travels along the interstitial bands of connective tissue to the pleural surface of the lung which ruptures. (2) The air in the mediastinum achieves sufficient tension to rupture the thin mediastinal wall into the pleural cavity. He cited the direct experimental evidence of Macklin[11] and considerable circumstantial clinical evidence to support the contention that the second route is the one usually taken. Our sixth case might prove to be a rather dramatic, direct, further clinical support of this contention.

CONCLUSIONS

1. Very little previous attention had been paid to pneumothorax as a complication of bronchoscopy when not dealing with foreign bodies.

2. We believe that pneumothorax will become a more frequently recognized condition attendant upon bronchoscopy as more frequent use of the fluoroscope occurs in conjunction with bronchoscopy.

3. Patients undergoing bronchoscopy have pulmonary disease which in itself may predispose to the occurrence of spontaneous pneumothorax.

4. Four examples of pneumothorax in conjunction with bronchoscopy and one case coincident to esophagoscopy have been presented. One case in which an extraordinary and fatal issue occurred subsequent to bronschoscopic examination is described. The true explanation of the course of events in this case remains a mystery.

5. We appreciate the fact that pneumothorax may follow direct trauma to the bronchial tree during bronchoscopy, but we feel that other factors produced the pneumothoraces described and these factors have been described in detail.

6. Pneumothorax has many serious dangers attendant upon it, and a consciousness of this complication in association with peroral endoscopy should prove lifesaving in some instances.

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EXPERIMENTAL HEMOTHORAX*

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INTRODUCTION

BLOOD coagulates in the pleural cavity. This fact, as true as it may be, has caused much controversy for over one hundred years. LeBlanc and Trousseau performed many experiments on horses (1829) and reached the conclusion that coagulation took place in the pleural cavity. Denny and Minot (1915), in experiments on dogs, reached the same conclusion. Elliott and Henry, during the period of World War I, presented both clinical and experimental evidence to prove that intrapleural blood coagulated. Sellors has reached the same conclusion from his experience in World War II.

The controversy has arisen because of persistent reports in the literature in which statements are made diametrically opposed to the findings of the authors just mentioned. Kato in particular has presented extensive and evidently fallacious evidence to show that the pleura elaborates an anticoagulant. With but few exceptions, most authors have based their opinions on clinical experience alone and confusion has resulted.

The purpose of this paper is to present experimental evidence in support of the theory that blood coagulates normally in the pleural cavity. An extensive review of the literature is also presented in order to bring the subject up to date.

REVIEW OF THE LITERATURE

Trousseau, in his lectures on clinical medicine delivered at Hotel Dieu, stated the following:

> Traumatic effusion of the blood is a pathological question in relation to which M. LeBlanc and I have made many experiments, the results of which have not received adequate publicity. The results of the experiments made by me and M. LeBlanc were performed in 1829 and published in the *Journal de Medicine Veterinaire* in 1834. We made a small incision in the skin of a horse between the middle-ribs; we carefully divided the tissues of the intercostal space and when we reached the pleura, we opened it in such a way as to avoid the lung and to limit the incision to some millimeters. By a stroke of the fleam, we then opened the jugular vein of the animal and then, by means of a kind of funnel, the small end of which was placed in the pleura and the other used to receive the product of the venesection, we introduced into the pleural cavity, one hundred, two hundred, four hundred, or even as much as three thousand grammes of blood. Having introduced the blood, we closed the wound by means of a twisted suture. In place of transmitting the blood directly from the jugular vein into the pleura, we generally received it in a syringe, and before it had time to coagulate, we injected it into the pleural cavity. We also divided an intercostal artery and allowed a certain quantity of blood to flow from it into the pleura.
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> The above experiment was performed on several horses. They were killed, some immediately after the operation and others after intervals from one, two,

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The original work on this subject was submitted by the senior author (D. W. M.) as a thesis in partial fulfillment for the degree of Doctor of Medical Science, [D.Sc.(Med.)], to the Faculty, Graduate School of Medicine, University of Pennsylvania.

Received for publication Aug. 25, 1945.

twenty-four, forty-eight and seventy-two hours, and from six to ten days. Without a single exception, however short was the interval between the injection and the autopsy, *we found the blood coagulated.* So rapidly did coagulation take place, that when, in our experiments, we opened an intercostal artery and caused the blood to flow directly into the pleural cavity, making at the same time an opening in a more dependent part of the chest, hardly a drop of blood flowed out of it. The same thing took place, when we injected from one to three kilogrammes of venous blood taken from the jugular vein, and at the time of injection, in a perfectly liquid state.

We repeated the following experiment several times. As soon as the injection was completed, we felled the animal by a blow on the head with a hammer. We then, without a moment's delay, opened the abdomen, exposing the diaphragm while the heart was still beating, and consequently, while physiological life was still quite preserved, I opened the pleural cavity through the diaphragm and found the blood in a clot. It was firmly coagulated, although the blood of the same horse taken at the same bleeding, but before the blood was injected into the pleura and left exposed to the air in an evaporating vessel, was only partially coagulated. Let me add that in cases in which the autopsy was made with the greatest possible celerity, not more than five minutes elapsed between injecting the blood into the pleura and ascertaining its condition there.

After a very short time, the serosity imprisoned within the clot partly bursts forth, and, *shaken up as it were by the movements of respiration,* is always mixed with a great quantity of blood globules. *At the first glance, it has the appearance of fluid blood.* There are two things then to be considered in cases of effusion of blood into the chest. There is the clot which generally occupies the most dependent parts—and there is the bloody serosity which comports itself exactly like the serosity of a pleurisy. If the surgeon were to tap, he would be able to withdraw a large quantity of serosity deeply colored by the cruor, and might thus come to the conclusion that he had withdrawn fluid blood! Surgeons then have not properly understood what takes place.

The lesion that has caused the effusion of blood into the pleural cavity will also cause an inflammation of the adjacent lung and pleura with a resultant outpouring of serum into the pleural cavity. We believe this to be the explanation rather than an inflammation of the pleura as a direct result of the irritation of the blood. We therefore contend that (1) blood in the pleural cavity coagulates, (2) it does not cause irritation, and (3) it is readily absorbed. As to contention No. 2, we have killed horses at four, six, and eight days after injecting blood into the pleura and never did we find evidences of pleurisy. As for contention No. 3, we have injected as much as 3,000 c.c. of blood into the pleural cavity and at the end of forty-eight hours determined that more than half of this was absorbed. After three days there remained only a small clot and a little reddish serosity.

Penzoldt (1876) stated: "Effusions of blood in the pleural cavity of mammals will remain liquid for a time—large effusions only a couple of hours—the smaller ones somewhat longer. The clotting is delayed by the healthy pleura. After more than twenty-four hours, however, the blood is always clotted. The fluid at the site of the clots was either the serum of the original blood and showed therefore no clotting after aspiration or, if it did show clotting, it indicated a secondary pleuritis."

Nelaton (1880), in experiments on dogs, found that blood injected into the chest resulted in a coagulum and a bloody serum. The serum looked like blood but was quickly resorbed. In some experiments where as much as 500 to 800 c.c. of blood were injected and examinations made on the fourth to the tenth day, there was found a great deal of noncoagulable bloody serum, but clots were present as well.[5]

Pagenstecher (1895), experimenting on dogs and rabbits, discovered that the blood remained almost completely liquid for two hours but that after six hours clots were always found and, in addition, a bloodlike, non-coagulable

fluid. He believed the pleura had the same power as the blood vessels in maintaining the blood in a liquid state. This coagulation in the pleura he likens to the process of thrombosis. Whereas thrombosis is caused by certain circumstances of trauma, so is the blood in a hemothorax influenced similarly.

Zahn and Walker (1914) found that blood that remained liquid in the pleural cavity (of dogs) did not coagulate in vitro either. Small amounts of blood (2 to 3 c.c.) did not clot. Large amounts of blood were found to coagulate always if the lungs were immobile. If the lungs were moving, the blood remained liquid in part. The coagulation of the blood could not be altered by means of pleural extracts. Exactly the opposite was found—when pulmonary tissue extracts were used, a more rapid coagulation occurred. The liquid pleural blood could always be coagulated by the addition of pure fibrinogen solutions. Therefore, the incapacity of the pleural blood to coagulate must depend upon an alteration of the fibrinogen. Failure of coagulation in the pleural cavity cannot be attributed to a fibrinolysin because fibrin is not dissolved in the pleural cavity. They felt that the pleural endothelium must in some way change the nature of the fibrinogen.

Denny and Minot (1915), in repeating Zahn and Walker's experiments, found the following: "Small amounts of blood introduced slowly into the pleural cavity when deep artificial respiration is maintained will remain in large part, fluid. In all cases, however, small clots were always found; the size of the clots depended upon the amount of blood injected and the rapidity of the injection. The fluid portion of the blood can be clotted only by addition of fibrinogen. Thrombin, calcium, and thromboplastin are incapable of causing coagulation. The blood shows an absence of fibrinogen. Pure fibrinogen which has been in the pleural cavity under the same conditions not only is not altered, but clots more readily than the control on adding suitable amounts of thrombin. Small amounts of thrombin and fibrinogen when mixed in suitable proportions and injected slowly into the pleural cavity resulted in a fluid portion and a clotted portion. There was an absence of fibrinogen in the fluid portion. Since it has been shown that fibrinogen alone loses none of its properties after remaining in the pleural cavity, and that the presence of thrombin under the same conditions causes a disappearance of fibrinogen, we can only conclude that coagulation has taken place. The experiments exactly parallel those performed with whole blood. The conclusion was reached that blood in the pleural cavity remains to a large extent fluid, not because of the presence of any "anti" substances, but because of coagulation, and defibrination produces a liquid that has all the gross appearances of whole blood but in reality is only serum and cells."

Elliott and Henry (1916) stated: "As blood flows from a wound into the pleural cavity, it clots rapidly. The clotting is not a massive coagulation such as one finds in a test tube of blood removed from blood vessels. The cardiac and respiratory movements agitate and 'whip' the blood during coagulation, so that its fibrin is thrown out and becomes deposited in layers of varying thickness on the parietal pleura and that part of the lung dipped into the effused blood. The throwing out of the fibrin leaves in the thoracic cavity a fluid which consists of blood serum together with practically all the usual cellular elements of the blood; that is, it is a fluid which has at this stage no power to clot because it contains no fibrinogen. Experiments have been performed to substantiate this contention. Later, after a period of time which varies greatly in individual cases, an inflammatory pleural exudate is thrown out and added to the pool of liquid blood. If much fibrin ferment is still present in the hemothorax after

primary clotting, the fibrinogen of this exudate will also be coagulated within the thorax and so increase the density of the fibrin enveloping the lungs. More frequently it escapes this change and then a sample of fluid taken from the pleural cavity exhibits a coagulation on standing which we term a secondary clot. This secondary clot has sometimes led individuals to the false conclusion that this is the primary clot in the ordinary effused blood which had never clotted within the pleural cavity."

Elliott (1919) stated the view of the coagulation process which he and Henry put forward was generally accepted, namely, that effused blood very rapidly passes through the clotting process.

Bradford and Elliott (1916) stated: "Blood aspirated from the chest any time after the fourth day may be of three different kinds. (1) A brownish red fluid, which yields a light gelatinous clot in vitro on standing and contains lymphocytes and endothelial cells. (2) A deep red fluid, exactly like nonclotted blood, which throws down a very heavy deposit of ordinary red cells on standing and may fail to give even the lightest clot in vitro. (3) Masses of soft blackish clot, like jelly, which soon blocks the needle. In two cases of hemothorax aspirated on the sixteenth day a dark-red fluid was obtained which clotted in vitro to a solid mass. At first the opinion was that in these two examples the blood had remained unclotted in the chest, while still retaining its normal coagulability on removal. Then it became necessary to admit that in example 3 the ordinary process of clotting must have already taken place in the chest, while in example 2, it was explained by the hypothesis of some peculiar change in the contrary direction which rendered the blood incoagulable. These contradictions were clarified by the findings of Elliott and Henry. Clotting in the pleura takes place always and very early through the action on the blood of the ferment liberated at the surface of wounded tissues. The clot in the chest may be (a) complete and massive, forming a soft and persistent clot such as found in example 3, or (b) massive, but with early and fairly extensive separation of the yellow serum from the clot as in example 1, or (c) interference with clotting by the churning movements of respiration so that fibrin is whipped out in layers which cover the pleural surfaces while the serum retains most of the red cells in suspension precisely as seen in defibrinated blood in a test tube. This last corresponds with example 2 and this fluid is found to be devoid of fibrinogen. The pleura reacts by a mild inflammation with a consequent emigration of endothelial cells, lymphocytes, and polymorphonuclear leucocytes with an outpouring of a variable amount of inflammatory fluid which contains fibrinogen and therefore yields a secondary clot outside the body when the fluid is allowed to stand. This clot may mimic closely a primary clot.

Gregoire and Courcoux (1919) stated that in an aseptic hemothorax in which the endothelial lining is practically intact, the blood does not coagulate but remains permanently incoagulable outside the pleural cavity.

Kato (1928), a Japanese worker, presumably confirmed experimentally that blood flowing into serous cavities remains either partially or completely unclotted. Thrombin, fibrinogen, hemoglobin, red cells, leucocytes, platelets, and calcium were reported as being diminished gradually while antithrombin increased. Extracts of the pleura contained large amounts of inhibitory substance. The strength of the inhibitory effect of the extract was parallel to the quantity of phosphorous present. This was attributed to an increase in nucleoprotein. It was concluded that the inhibitory substance in the thoracic blood, though spoken of as "antithrombin," remains unidentified, its true nature and mode of action

unexplained. It appeared, however, that the activity of this "anti" substance was in some way related to the amount of phosphorus present.

Filatov (1928) stated the failure of coagulation was due to a selective biochemical reaction between the blood and the secretion of the serous lining of the pleural cavity which resulted in destruction of fibrinogen.

Hopkins (1937) stated: "It has long been known that blood in the pleural cavity shows little tendency to clot."

Foster (1938) stated: "The explanation of Elliott and Henry of blood coagulation in the pleural cavity is not tenable because the blood coagulates immediately after being withdrawn from the pleural cavity."

Tannenbaum (1942) stated: "Blood escaping into the pleural cavity causes an effusion. The effusion presents the blood from clotting because it undoubtedly contains anticoagulants.

Edwards (1943) stated: "Extensive clotting is rare in the pleura though it is common to see smaller clots in the costophrenic angle. No adequate explanation has as yet been put forward to account for this."

Nicholson and Scadding (*Lancet*, 1944) stated: "Clotting in a hemothorax is rare."

In an editorial in the *Lancet* (1944) it was stated that: "Blood in the pleural cavity rarely clots."

d'Abreu stated in the *Lancet* (1944): "Clotting, if it does take place, is due to infection."

Carter and Debakey (1944) stated: "Clotting of blood in the pleural cavity occurs rarely and the mechanics responsible for this phenomenon was not entirely clear."

Bigger stated: "Aspiration may prevent the formation of clots in hemothorax."[52]

Van Allen stated: "The pleura is known to have a powerful anticoagulant property so that freshly drawn blood poured over the pleural surface does not coagulate."[44]

Alexander (1944) stated: "Blood in the pleural cavity tends to remain fluid."

Barrett (1945) stated: "The blood does not clot immediately and all the factors which influence this process is not certainly known. It tends to remain fluid for a long time."

Price and Cleland (1945) stated: "As far as we know, no satisfactory explanation of the occasional occurrence of clotting, or perhaps, more significantly the usual absence of clotting has been put forward. The etiology of massive clotting obviously requires further careful investigation, particularly of a biochemical nature for its elucidation."

Coleman (1945) made the statement that blood in the pleural cavity apparently clots rapidly but made his statement on the findings of Elliott to whom he refers.

Sellors (1945 stated): "In hemothorax clotting occurs at ordinary speeds."

Tuttle, Langston, and Crowley (1945) stated: "In early thoracotomies done for hemothorax alone. the blood within the pleural space was not clotted in all instances."

THORACIC ANATOMY OF THE DOG

Attention is directed to the anatomy of the thorax in the dog because of the difference of opinion regarding the division of the right and left cavities into separate compartments. It is a common statement of investigators using

the dog as the experimental animal that fluid and air readily find their way from one pleural cavity to the other no matter on which side the injection is made. This has led a number of investigators to presume there must be an actual anatomic defect between the right and left pleural cavities to allow such a passage. Other individuals offer no explanation except to say that for all practical and experimental purposes, the thorax of the dog must be considered to be composed of a single cavity. Graham, as a counter argument to those who criticized his work on empyema, examined twenty-five dogs with the specific purpose of determining the existence of an anatomic connection between the right and left pleural cavities. He could not find one. He did agree that the mobile mediastinum was so easily displaced that any intrapleural change on one side was reflected in the opposite pleural cavity in such a manner as to make the two pleural cavities function as one. In many of the dogs used in the following experiments, blood injected in the right pleural cavity was found to pass readily to the left side. This led naturally to investigation as to the mechanics of this transfer. It became evident that a definite anatomic connection did not exist as Graham had previously declared. The best description of the anatomy of the pleural cavity in the dog was found to be Bradley's: "If the parietal pleura is traced in a ventral direction, it will be found to reach the middle line of the sternum where it meets its fellow membrane of the opposite side. From the sternum each membrane is reflected dorsalward as the mediastinal pleura. Close to the sternum the two pleurae are intimately related and a thin double membrane is produced. On the right side of the thorax the arrangement of the pleura is complicated by the presence of a fold that leaves the diaphragm and passes dorsalward to surround the caudal vena cava and the right phrenic nerve. The mediastinum is divided into three parts: (1) A precardial mediastinum cranial to the heart, (2) a cardial mediastinum containing the heart and other structures occupying the same transverse zone of the chest, and a (3) postcardial mediastinum of triangular outline and circumscribed by the heart, diaphragm, and vertebral column. The precardial and cardial mediastinum are in a median position but the postcardial mediastinum is pushed to the left by the accessory lobe of the right lung as it passes underneath the caudal vena cava."

In postulating the mechanics of transfer it was quite apparent that any slight amount of trauma might easily rupture the mediastinal septum. The septum, particularly in the ventral substernal location, is tissue-paper thin. The heart is suspended in this septum and can be shifted deep into either the right or left pleural cavities with ease. When fluid or air is introduced into either cavity, the heart shifts and this shift plus the pull exerted by the weight of the heart causes a defect of the ventral substernal mediastinal septum. It is through this traumatic defect that transfer of fluid and air is thought to take place.

Referring again to the anatomic description of Bradley, another interesting anatomic variation is the infracardiac "pocket." This pocket (described by Bradley as the triangular outline of the postcardial mediastinum) contains the accessory lobe of the right lung. Elkin stated at one time that this was an empty collapsed serous cavity that exhibited itself as a pocket when the chest was opened. In a number of observations, the accessory lobe of the right lung was found to pass below the inferior vena cava on the right and come to rest in this pocket just as described by Bradley.

In summary, the investigations relative to the anatomy that have been carried out in this study add nothing to the excellent description of Bradley. They do corroborate the fact and thereby substantiate the contentions of Graham and there is no actual anatomic communication between the right and left pleural cavities in the dog. Furthermore, the postcardial mediastinal pocket serves a very definite function as it harbors the accessory lobe of the right lung and is not a potential collapsed space as Elkin thought.

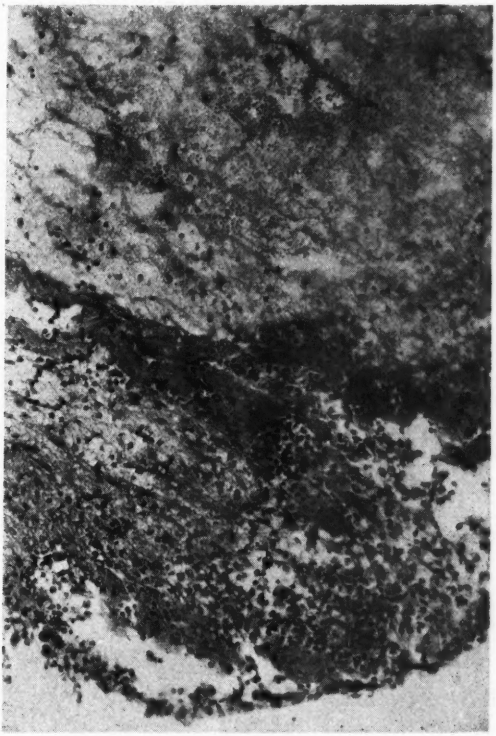

Fig. 1.—Blood clot in the pleural cavity (forty-eight hours). The blood had been in place in the pleural cavity for forty-eight hours. Note the fibrin and enmeshed erythrocytes at the top of the illustration. It is adherent to the mediastinal septum shown at the bottom. Note the cellular infiltration of the septum.

METHOD AND RESULTS

The method used was an indirect transfer of blood from a donor dog to the pleural cavity of the recipient dog by a multiple syringe method. Blood was taken from the femoral artery of the donor in a syringe that had been previously moistened with 3 per cent potassium oxalate or mineral oil. By using the femoral artery, it was possible to obtain 100 c.c. amounts of blood rapidly. Transfer to the pleural cavity could thereby be carried out with a minimal amount of time elapsing between drawing the blood and injection. It was recognized early that speed was necessary in transfer of the blood because of the rapid coagulation of dogs' blood. Oxalate or mineral oil was used with the idea of preventing the barrel of the syringe from sticking. Neither had an effect on the coagulation time of the blood as determined by estimation of the prothrombin time. Other methods at first tried were not successful because

of the difficulty, as a result of the coagulation, in getting large amounts of blood rapidly. It is, therefore, of more than passing interest to make note of the method for obtaining large amounts of blood rapidly. This is done by isolating the femoral artery and freeing up a one-inch section by sharp and blunt dissection. The distal portion of the femoral artery is then grasped in a Kelly hemostat and the aspirating needle inserted into the artery just proximal to the hemostat. The hemostat thus acts as a point of fixation of the artery and facilitates the arterial puncture. After the desired amount of blood is withdrawn, a second Kelly hemostat is used to close the hole made by the first puncture. In this way the femoral artery can be punctured as many as five times with ease. If the dog is a large one, 500 c.c. of blood can be obtained with a minimal amount of difficulty. The high pressure in the artery is, of course, the secret of rapid withdrawal of blood.

Fig. 2.—Blood clot in the pleural cavity (five days). The blood clot is shown in the lower right of the illustration. Note again the cellular infiltration of the mediastinal septum. In the center the fibroblastic proliferation is quite evident.

To avoid repetition some of the experiments have been grouped. Experiment 10 is representative of the first ten experiments performed with the exception of the time interval between injection of the blood and examination of the pleura. The conclusions, therefore, are a condensation of the results obtained in the total of ten experiments. Experiment 14 is illustrative of one of a group of nine identical experiments. Single experiments have been reported in detail where their importance justifies it. Inspection of the pleural cavities was carried out at intervals varying from ten minutes to forty-six days. Coagulation of the blood was an invariable finding and is illustrated by sec-

tions taken of the residual clot at forty-eight hours, five days, and forty-six days (see Figs. 1, 2, 3).

Experiment 10.—The following is presented as typical of the first ten experiments. The dog was used as the experimental animal.

Three hundred cubic centimeters of whole blood were taken from the femoral artery and introduced into the pleura. Forty-eight hours later the pleural cavity was examined. There were two very large clots in the right pleural cavity (side of injection), a medium-sized clot in the left pleural cavity, and another large clot in the postcardial mediastinal pocket. There was fluid blood in both pleural cavities. The mediastinal septum was reddish in appearance. Some of the clots were tightly adherent to this septum. Clots not in contact with the septum failed to adhere to the visceral or parietal pleura. Microscopic sections were made of the septum and adherent clot. Figs. 1, 2, and 3 are photomicrographs to illustrate clot and septum. A substernal lymph node was removed and sectioned. Intact erythrocytes were readily recognized within large macrophages (see Fig. 4). Examination of the septum failed to reveal similar macrophages containing engulfed erythrocytes. Positive evidence is therefore lacking to allow postulation that macrophages carried the red cells to the lymph node by way of the mediastinal septum. Higgins and Lemon studied the activity of the macrophages in the mediastinal septum and were unable to demonstrate the presence of lymphatic channels.

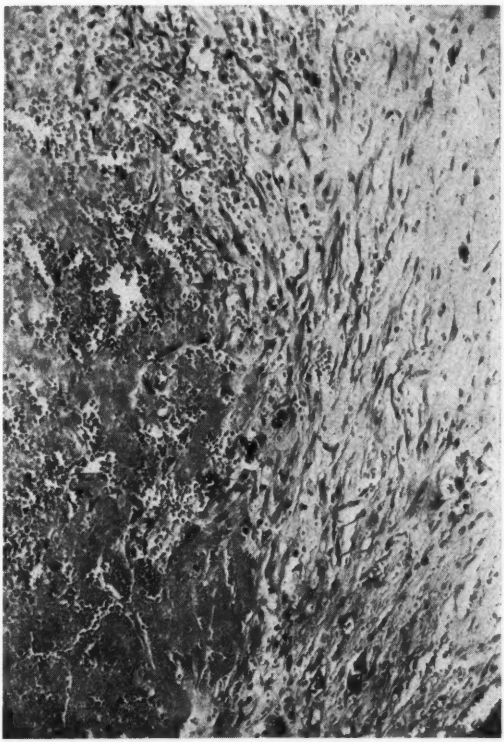

Fig. 3.—Blood clot in the pleural cavity (forty-six days). The blood clot is seen on the left. The mediastinal septum on the right shows a decreased cellularity when compared with Figs. 1 and 2. Note the fibroblasts infiltrating the clot.

The fluid portion of the blood was examined. One specimen allowed to stand in a test tube at room temperature remained indefinitely incoagulable. A prothrombin time determination was carried out on another specimen with the result that no clot formed. Smears were made of this fluid blood and they were examined for red cells and platelets. Normal-appearing erythrocytes were present, but platelets were consistently absent.

Comment.—The important findings of this first group of experiments may be condensed as follows:

1. Blood will clot in the pleural cavity. Both the liquid portion of the blood and the clot are absorbed readily. The liquid portion will disappear within ten to fourteen days and the clot will have decreased in size to such an extent that it may easily be overlooked. Representative clots removed at forty-eight hours, five days, and forty-six days are illustrated by Figs. 1, 2, and 3. After

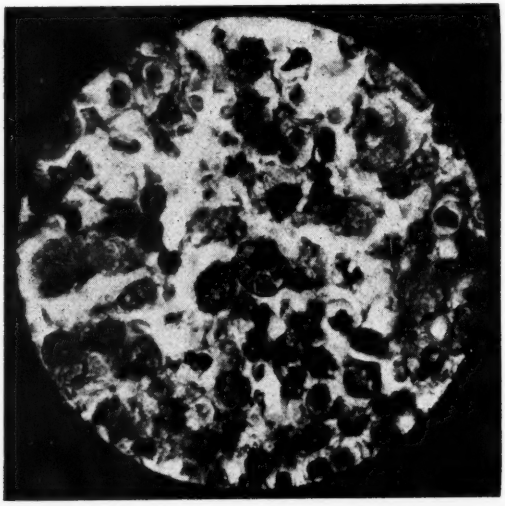

Fig. 4.—Absorption of blood from the pleural cavity (forty-eight hours). A mediastinal (sternal) lymph node. Note the macrophages engorged with erythrocytes. Examination of the mediastinal septum of the same dog failed to reveal macrophages which contained erythrocytes. The question then arose as to how the red cells reached the lymph node. Lemon and Higgins failed to demonstrate lymphatics in the mediastinal septum. If lymphatics could be demonstrated, then one might account for the transfer of the red cells in the macrophages to the lymph node via the septum. Lacking these lymphatics, then, the method of transfer is as yet unanswered. The experimental investigation of this problem is not included in the present paper.

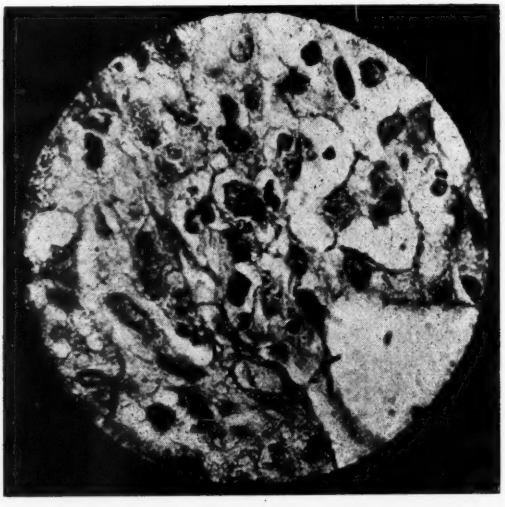

Fig. 5.—Absorption of blood from the pleural cavity (forty-six days). The mediastinal septum after blood had been in place in the pleural cavity for forty-six days. The macrophages here are engorged with hemosiderin. The hemosiderin imparted a brownish stain to the mediastinal septum.

two weeks have elapsed, there remains little evidence that blood had previously been present in the pleural cavity. Small clots and a brownish staining of the mediastinal septum will be the only residuals. The visceral and parietal pleura remain clean and glistening.

2. The red cells are not laked immediately after introduction into the pleural cavity. They are recognizable after five days, although some are fragmented at that time. The fragmented erythrocytes are taken up by the macrophages of the septum and appear there as hemosiderin within the phagocyte (see Fig. 5). Adjacent lymph nodes reveal large macrophages containing phagocytosed intact erythrocytes (see Fig. 4).

3. The liquid portion of the blood will not clot. Prothrombin time determinations fail to result in a clot. Platelets rapidly disappear from blood introduced into the pleural cavity.

4. An actual anatomic communication cannot be demonstrated between the two pleural cavities. This is simply corroborative evidence to the findings of Graham. The postcardial mediastinal pocket is peculiar to the thoracic anatomy of the dog. It is recognized as the receptacle for the accessary lobe of the right lung.

Experiment 14.—It had been observed in the preceding experiments that blood withdrawn from the pleural cavity failed to clot even upon standing several days. In order to determine which of the necessary coagulation substances was missing, the following series of tests were run: Blood was drawn into either oiled syringes or syringes wet with potassium oxalate. It was then placed into the pleural cavities of both living and dead dogs and allowed to remain for varying lengths of time. When the chest cavities were opened, clots were found. In those dogs in which respiratory excursion continued (the live dogs), a large part of the blood remained liquid. In those dogs in which respiratory excursion had been abolished (the dead dogs), there was a small amount of "fluid" blood and a large clot. Prothrombin determinations were run on samples of the "fluid" blood using Pohle and Stewart's modification of the Quick procedure. In all cases clot formation failed to occur. From these results it was concluded that prothrombin or fibrinogen was absent. It is to be noted that the one-stage method depends on an adequate concentration of fibrinogen. In fibrinogenopenia, the "prothrombin time" would naturally be prolonged.

Citrated blood was then introduced into the pleural cavities of both living and dead dogs. After a reasonable length of time (from fifteen to twenty minutes), the chest cavities were opened and samples obtained. No clots were noticed in this series of animals. Prothrombin time determinations on these samples were found to be normal, that is, from six to seven seconds. Plasma from these samples was also recalcified, that is, plasma from the citrated blood which had not been introduced into the pleural cavity, and no significant difference was noted between these and the controls. Thus it was shown that blood previously decalcified underwent no change when placed in contact with the pleura. This was evidence against the idea of an anticoagulant being secreted by the pleura. It also shows that none of the substances necessary for coagulation were adsorbed on to the pleura to any significant degree.

In order to determine exactly which constituent necessary for coagulation was absent from the uncitrated blood, the following experiments were performed: Specimens of blood, both citrated and noncitrated, were placed in the pleural cavities of a series of dogs. One-tenth cubic centimeter of thrombin* containing either 100 units or 20 units was added to 0.1 c.c. of plasma and 0.1 c.c. of .005 M. calcium chloride. In all samples of citrated plasma, clotting was practically "instantaneous" as would be expected. Plasma from the noncitrated samples failed to clot when thrombin and calcium chloride were added. These experiments showed that thrombin or prothrombin was not the missing factor. The following system was then set up: 0.1 c.c. 10 per cent fibrinogen* plus 0.1 c.c. .005 M. calcium chloride plus 0.1 c.c. plasma obtained from the non-citrated blood removed from the pleural cavity. Clot formation took place. This experiment was repeated a number of times. In one experiment,

*The thrombin and fibrinogen were obtained from Dr. W. H. Seegers, Parke, Davis & Company, Detroit, Mich.

a weaker solution (1 per cent) of fibrinogen was used. Clot formation failed to occur. Occasionally, when it was found that fibrinogen failed to clot the "plasma," technical details were in error. These results are summarized in Table I. It is to be noted that in all of the experiments in which technical difficulties were not encountered, the 10 per cent fibrinogen solution consistently caused clot formation. The clotting time varied between one and three minutes in a series of seven dogs. The interpretation of these data was that thrombin was present in the fluid portion of the blood and hence this fluid portion must in reality be serum. Thus, we came to the conclusion that blood placed in the pleural cavity clotted in the usual manner. Clots formed in the living animal were small due to the mechanical action of respiration. Clots formed in the dead animal were larger and the fluid portion smaller. The fluid which is apparently noncoagulated blood is then, in reality, serum with suspended red cells or defibrinated blood.

TABLE I. ACTION OF THROMBIN AND FIBRINOGEN

	ARTERIAL BLOOD	INTRAPLEURAL BLOOD*			
		(1)	(2)	(3)	(4)
	CONTROL CITRATED PLASMA	LIVE DOG CITRATED PLASMA	DEAD DOG CITRATED PLASMA	LIVE DOG UNCITRATED "PLASMA"	DEAD DOG UNCITRATED "PLASMA"
Prothrombin times .1 c.c. plasma + .1 c.c. thpl† + .1 c.c. $CaCl_2$	7″	6″	7½″	No clot	No clot
Recalcification .1 c.c. plasma + .1 c.c. $CaCl_2$ (.005 M. $CaCl_2$: end point = inversion of tube)	3′	2′	3′		
Thrombin .1 c.c. thrombin + .1 c.c. $CaCl_2$ + .1 c.c. plasma	Instant clotting	Instant clotting	Instant clotting	No clot	No clot
Fibrinogen .1 c.c. fibrinogen + .1 c.c. $CaCl_2$ + .1 c.c. "plasma" (typical experiment)				3′	2′

*(1) and (2): The blood was citrated as it was withdrawn from the femoral artery. This citrated blood was then used for intrapleural injection. Following its removal from the pleural cavity it was centrifuged to obtain the plasma.

(3) and (4): The blood was aspirated into oiled syringes and immediately injected into the pleural cavity. Following its removal from the pleural cavity, it was centrifuged to obtain the "plasma." *Note* that the word plasma is placed in quotation marks. This is done to point out our contention that clotting has taken place; fibrinogen is missing, and therefore, it is in reality serum and not plasma.

†thpl, Thromboplastin; $CaCl_2$, .005 M.

Experiment 19.—

Dog 34: The dog was anesthetized and then an endotracheal tube was inserted in order to maintain positive pressure anesthesia. The chest was opened and the lung rhythmically inflated under positive pressure in order to keep the dog alive and also in order to simulate the movement of the normal lung. Blood was slowly dripped over the moving lung. Other individuals have claimed that such a procedure will result in the blood staying liquid because of the anticoagulant action of the visceral pleura. To the contrary, here, a very thin, but definite film of coagulated blood formed where the blood came in contact with the visceral pleura.

Experiment 12.—It was thought that if the pleural endothelium did elaborate an anticoagulant, the transfusion of pleural blood should cause a change in the prothrombin time of the recipient dog.

Two hundred fifty cubic centimeters of blood were removed from the femoral artery of Dog A and injected into the pleura of Dog B. Dog B was kept alive until 120 c.c. of the liquid intrapleural blood could be removed by thoracotomy and transfused back into Dog A. This transfusion was rapidly performed. It was completed in ten minutes and the first blood specimen on Dog A was taken three minutes after the completion of the transfusion. Specimens of blood were then drawn from Dog A at fifteen-minute intervals and prothrombin times determined. The transfusion of the pleural blood had no effect on the prothrombin times whatsoever. The prothrombin times were all normal.

Different dilutions of the pleural blood were mixed with whole blood and no anticoagulant effect of the pleural blood was noted. The blood coagulated normally.

Experiment 13.—In order further to disprove the anticoagulant action of the pleural endothelium, the following was done: A six-inch square of parietal pleura was dissected away from the thoracic cage by separation of the pleura in the plane of the endothoracic fascia. This piece of parietal pleura was then cut in small pieces with a scissors, placed in a mortar, macerated with a pestle, and 30 c.c. of 1 per cent normal saline were slowly added. After thorough maceration the specimen was placed in a test tube and centrifuged. The supernatant fluid was tested for anticoagulant activity (see Table II). The contrary was found. The fluid thus obtained from maceration of the pleura resulted in a hastening of coagulation. In other words, it had a thromboplastic action and not an antithrombic action. Kato, a Japanese investigator claimed that extracts of the pleural endothelium made as described exerted a profound anticoagulant action.

TABLE II. SYSTEMS USED TO TEST ACTIVITY OF PLEURAL EXTRACT

	ARTERIAL BLOOD	INTRAPLEURAL BLOOD*	
	CITRATED PLASMA CONTROL	CITRATED PLASMA DEAD DOG	CITRATED PLASMA LIVE DOG
Prothrombin time	6½″	6½″	5½″
Recalcification	23½″	40″	109″
.1 c.c. plasma + .1 c.c. .85% NaCl + .1 c.c. .005 M. $CaCl_2$			
Pleural extract (P.E.)	7″	7½″	
.1 c.c. plasma + .1 c.c. thpl† + .1 c.c. P.E. + .1 c.c. $CaCl_2$			
.1 c.c. plasma + .1 c.c. thpl + .1 c.c. NaCl + .1 c.c. $CaCl_2$	8″	7½″	
.1 c.c. plasma + .1 c.c. P.E. + .1 c.c. $CaCl_2$	10½″	12½″	

*The blood was citrated as it was withdrawn from the femoral artery. This citrated blood was then used for the intrapleural injection. Upon its removal from the pleural cavity it was centrifuged to obtain the plasma.

†thpl, Thromboplastin.

Conclusion: A saline extract made from macerated pleural endothelium does not have an anticoagulant action.

Although pleural absorption is not the main interest of this paper, the routes by which the formed blood elements are removed from the pleural cavity are as yet unsolved and the following summary of the situation is in order. As stated previously, blood is absorbed from the pleural cavity quite readily. One might be tempted to believe that the mediastinal septum is responsible for this through the agency of phagocytic macrophages and lymphatics.

Phagocytosis occurs but this does not seem to be an adequate explanation for absorption of blood. Phagocytosis is a slow process and, for the most part, the absorption is accomplished rapidly. Furthermore, Higgins and Lemon were unable to show that the mediastinal septum contained lymphatic channels. They found graphite particles were anchored in the mediastinal septum for long periods of time. This proves a stumbling block if one desires to ascribe any great powers of absorption to the mediastinal septum. How then does one explain absorption from the pleural cavity agreed that it is a rapid process? Starling is given credit and Rountree later corroborated the fact that any substance in solution will be absorbed from the pleural cavity by way of the blood stream. For an explanation of rapid removal of particulate matter, we must refer to Allen's works on the peritoneum. In brief, he explains the removal of red cells from the peritoneal cavity on direct ingress of the erythrocytes to the lymphatic channel by way of peritoneal stomas. A schematic drawing of his concept of the lymphatics of the diaphragm is shown in Fig. 6. In Fig. 7 is a reproduction of a photograph showing the passage of erythrocytes through the stomas into the lumen of the lymphatic. This concept takes us back to that of von Recklinghausen. Many have denied and some supposedly

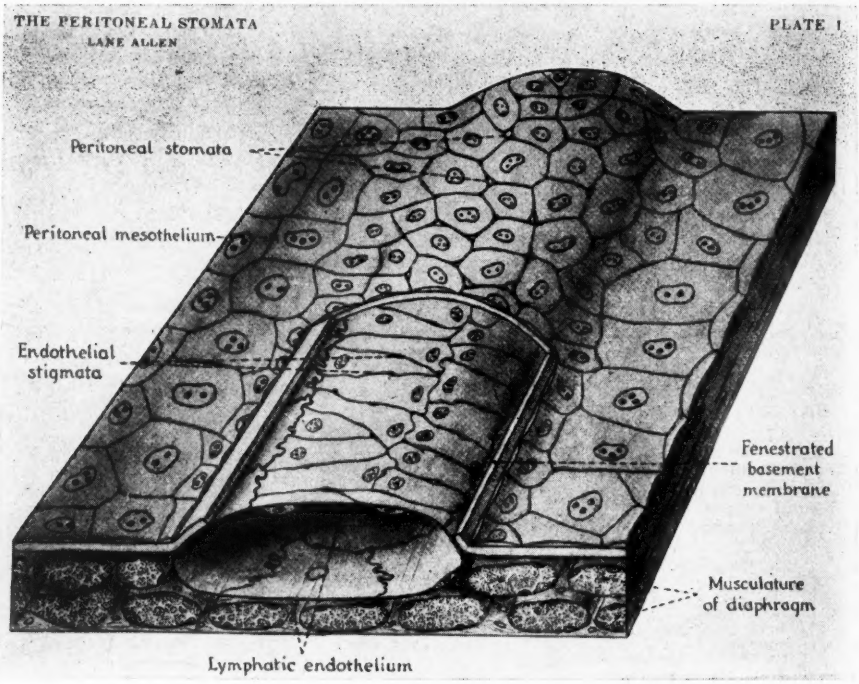

Fig. 6.—A cross section of the diaphragm illustrating the stomata overlying the lymphatic vessel. The concept of von Recklinghausen has been re-introduced in order to explain absorption from the peritoneal cavity. The same method may or may not be at work in the pleural cavity. It is not the purpose of this paper to prove or disprove such a method of absorption. It does appear unavoidable, however, that some mechanical means must be advanced to explain the greater part of absorption from the pleural cavity. (From Allen, L.: Anat. Rec. 67: 89, 1936.)

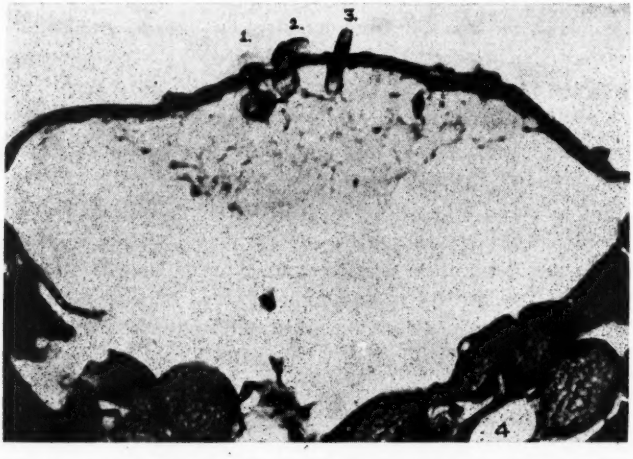

Fig. 7.—Represents a red cell entering the lymphatic vessel by way of the peritoneal stomata. (From Allen, L.: Anat. Rec. 67: 89, 1936.)

proved (Miller and Hertzler, in particular) that these stomas of von Recklinghausen were nothing more than artefacts. Florey described the passage of graphite out of the peritoneal cavity between the mesothelial cells as shown in Fig. 8. He thought intra-abdominal pressure forced the particular matter between the cells. It is only by *inference* from these workers with reference to

absorption from the peritoneal cavity that one can reason that a similar method of absorption is at work in the pleural cavity. In general it may be stated that absorption from the pleural cavity probably takes place as follows: Substances in solution will be taken up by the blood stream and particulate matter will be removed by the lymphatics either by way of phagocytosis or by direct ingress of the particulate matter to the lymphatic channel. Probably the conclusion given by Levi regarding the foregoing is worth while repeating: "I am inclined to believe that the immigration of cells (from the peritoneum) is a mechanical process, but as to the exact mechanism, I am still in doubt." A similar statement is equally applicable to absorption of blood cells from the pleural cavity.

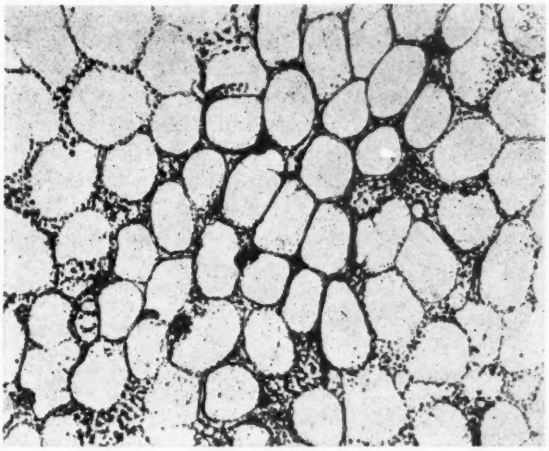

Fig. 8.—Represents a drawing of the surface of a rat's diaphragm showing the outlining of the mesothelial cells after intraperitoneal injection of hydrokollag 300. Florey does not think there are peritoneal stomas and uses this drawing to illustrate his idea of absorption from the peritoneal cavity. (From Florey, Howard: Brit. J. Exper. Path. 8: 479-489, 1927.)

DISCUSSION

The foregoing experiments, it may be seen, closely parallel those of Denny and Minot and the agreement in experimental findings is worthy of note. They also agree with the experiments of Elliott and Henry, and nothing has been added to the original contentions regarding changes of blood in the pleural cavity. A recent experiment closely related to the problem of coagulation is that of Lozner, Taylor, and Taylor (1944) on the coagulation defect in menstrual blood in which they concluded that the fluidity of the menstrual blood is caused by the absence of both prothrombin and fibrinogen. Expressed in simpler language, it follows that menstrual fluid is blood that has already clotted and that there is no "coagulation defect." The same is true of hemothorax.

Assuming the experimental evidence herein presented to be accurate, then we immediately become concerned with its application to hemothorax in human beings. In order to link the experimental to the clinical, it is fortunate we can present the findings of Sellors. His studies deal exclusively with hemothorax in human beings. His article will bear repeating in some detail in order to make clear the agreement between the experimental and the clinical findings.

Sellors (1945) stated: "It is now recognized that clotting does occur at ordinary speed, but owing to the physiomechanics of the chest, the hematoma is rapidly defibrinated so that the actual gel form of clot is transposed into shreds of fibrin floating in a pool of liquid blood. The gross disturbance in the

physiomechanics caused by the disturbance of the intrathoracic organs plus the blood loss results in increased pulse rate, increased respiratory rate, and (in sucking wounds) a mediastinal flutter resulting in violent agitation. Any clot that forms has little chance of remaining static and is rapidly broken up and fragmented. The impression (when the chest is opened soon after injury) is that of a collapsed lung splashing around in a pool of blood and bouncing with each transmitted pulsation of the heart. It is easy to understand how defibrination occurs.

"When the hemothorax fluid has been removed a small residue of clot or fibrin may be found in the depths of the cavity but in some instances little solid matter can be identified even on careful inspection. Best results are obtained when cases were operated upon within a few hours of injury. In these cases the whole volume of fluid was sucked out and thoroughly mixed before determinations were made on the specimens. The hemoglobin content of the hemothorax was from 30 to 40 per cent below that of the circulating blood and the fibrinogen content was virtually nil. The complete evacuation of the chest under direct vision should dispose of the contention that a deposit of fibrin would carry down an undue proportion of red cells. In one case the volume of fibrin was only 60 Gm. in over 1,500 c.c. of liquid blood. Herewith is presented the findings in this case of hemothorax operated upon and emptied within six hours."

	HEMOGLOBIN (%)	PROTEINS	FIBRINOGEN (GM./100 C.C.)
Normal blood	100	6 to 7.5	0.2:0.45
Venous blood (case in question)	94	6.4	0.41
Pleural fluid (case in question)	46	5.5	0.025

The details of a second case of hemothorax are illustrated in Fig. 9; treatment by aspiration.

In Fig. 10 are summarized the findings of Sellors. Particular note is called to the number of determinations (143 readings in eighty-six cases in Fig. 10*A* and 121 readings in seventy-one cases in Fig. 10*B*).

"The reduction of hemoglobin, even allowing for error, suggests there was some dilution of the fluid by a reactionary pleural exudate. The effusion probably produces its greatest effect on the third day with signs and symptoms of intrathoracic tension with respiratory and mediastinal displacement. This is sometimes interpreted as due to further active hemorrhage and prevents the clinicians from removing more than a small amount of the total fluid. The fear of restarting bleeding is groundless.

"In a properly treated patient the pleural cavity is dry by the fifth or sixth aspiration. In most of the patients subject to routine aspiration, the pleural cavity was dry by the eleventh to fourteenth day; if continuity of treatment were possible, this should be accomplished around the seventh to tenth day.

"The virtual absence of fibrinogen is noted in the early stages and thereafter the percentage rises until in some patients it is higher than the blood level after one week or more. It may be inferred that an active serous exudate is produced. The conversion from sol to gel is seen in the later aspirations, while it is not seen in the early aspirations or otherwise when the fibrinogen content is low. The significance of an exudate lies in the fact that a considerable quantity of fibrin may be deposited on the walls of a hemothorax cavity. The greater amount of deposit (up to one inch) is found on the parietal pleura in comparison to the visceral pleura (average, one-eighth inch).

"The essence of recovery from a hemothorax is that the lung should reach the chest wall and obliterate any pleural cavity at the earliest moment. If fibrin develops before the pleural layers become apposed, the closure is mechanically delayed. Once the fibrin has organized, it acts as a constricting barrier and encourages persistence of dead space. The risk of fibrothorax develops. This means full respiratory function is not recovered. This is the inevitable state of affairs in hemothoraces of appreciable size which are treated conservatively."

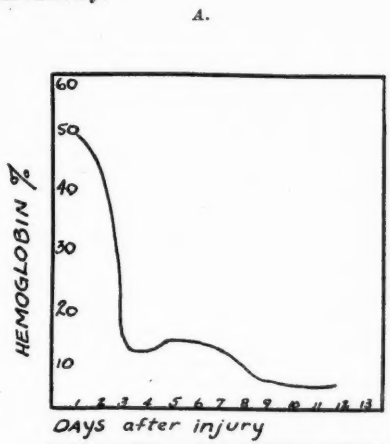

Fig. 9.

Fig. 10.

Fig. 9.—Second case of hemothorax; treatment by aspiration. (From Sellors, T. H.: Lancet 1: 143-144, 1945.)

Fig. 10.—*A*, Hemoglobin content of fluid aspirated from hemothoraces. *B*, Fibrinogen content of fluid aspirated from hemothoraces. (From Sellors, T. H.: Lancet 1: 143-144, 1945.)

An editorial in the Lancet followed the above article: "The treatment of hemothorax then is no longer a conservative procedure aimed only at preventing infection. It should be early evacuation of pleural contents and vigorous attempts to restore full lung expansion by breathing exercise. Even slight delay in evacuating the pleural cavity may incapacitate a patient for months instead of just a few weeks."

CONCLUSION

LeBlanc and Trousseau in 1829 performed experiments on horses to show that blood coagulates normally in the pleural cavity. Denny and Minot in 1915 and Elliott and Henry in 1916 reached the same conclusion. These workers

found fibrinogen was removed from the pleural blood. This indicated to them that the coagulation process had gone to completion. By adding fibrinogen to the liquid portion of the pleural blood they were able to cause clotting. During the interim 1916 to 1945 there has been a good many controversial statements made regarding the coagulation of blood in the pleural cavity. Experiments have been reported (Kato, 1928) to show that an antithrombin is formed in the pleural cavity which prevents coagulation. Other individuals have expressed opinions regarding the "noncoagulation" of blood in the pleural cavity. Most of these opinions are irrelevant as they are unsubstantiated ideas of each individual author. Experiments are herein reported to add corroborative evidence to the work of LeBlanc and Trousseau, Denny and Minot, and Elliott and Henry. Blood introduced into the pleural cavities of dogs coagulates normally. The motion of the chest will alter the physical appearance. There will be a liquid portion (serum and cells) and a solid portion (fibrin and cells). The liquid portion, being nothing more than serum and cells, will remain indefinitely incoagulable. Fibrinogen added to this liquid portion will bring about coagulation. Additional experiments are also herein reported to prove that the pleura does not elaborate an anticoagulant. This is, therefore, the opposite of Kato's findings. The experiments in general show that the pleural cavity readily absorbs blood.

The experimental studies were carried out under the supervision of Dr. E. R. Schmidt, Professor of Surgery, University of Wisconsin School of Medicine. We are indebted to Dr. W. J. Meek, Dr. O. O. Meyer, and Dr. A. R. Curreri for help and suggestions. In particular we appreciate the assistance given us by William Kaplan and Wilbert Heimann.

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NERVE BLOCK IN THE TREATMENT OF THORACIC INJURIES

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THE importance of therapeutic nerve block in the early treatment of thoracic injuries is well established. Nerve block is effective in injuries which vary from simple uncomplicated chest wall contusion to lesions with compound, comminuted fractures of the ribs, including "flail" chest. Regardless of the size or type of wound, pain usually is the predominant symptom. With relief of pain, patients will respond better to shock therapy. In addition, they withstand transportation better and they travel with greater ease and comfort. Nerve block is more physiologic and far more efficient than chest strapping in controlling this pain, because the strapping favors anoxia and retention of bronchial secretions by limiting pulmonary expansion and interfering with the cough mechanism.

Not infrequently thoracic pain may be accompanied by a moist, ineffectual cough, dyspnea, and, occasionally, cyanosis—the "traumatic wet lung" syndrome. There are diminished breath sounds, moist bronchial râles or wheezes, and limitation of motion on the affected side. The râles are more marked on the injured side but often they may be present on the contralateral side. The relief of pain permits the patient to cough and to raise blood and secretions which are hindering pulmonary ventilation. The clinical response is often dramatic. The dyspnea abates, the cyanosis disappears, and the general condition of the patient is greatly improved. The immediate response is due primarily to the relief of pain, but it may be due in part to the relief of the bronchial and bronchiolar spasm.[1]

A painful chest may be relieved by the local infiltration of procaine at the site of injury[2] or by an intercostal or paravertebral nerve block.[3] However, in lacerated and contaminated wounds, local infiltration at the wound site is contraindicated. This leaves the intercostal or the paravertebral block as the method of choice. The total number of intercostal or paravertebral injections to be made is determined by the extent of the lesion and the subjective relief of pain. Because of its simplicity the intercostal nerve block is used as frequently as possible; however, if this procedure does not prove satisfactory, or, if the site of injury is located over the angle of the rib, a paravertebral block is used. If the site of injury is located so posteriorly that even a paravertebral block will mean injecting through the contaminated area, a paravertebral block incorporating at least two nerve segments above the superior margin of the lesion may be of aid in blocking an appreciable number of the pain impulses which travel cephalad in the paravertebral sympathetic chain. Generally speaking, the relief of pain following a successful block persists for a minimum of two to four hours, and it is rare that more than three consecutive blocks must ever be performed. In our experience, persistent relief following one effective block is the rule rather than the exception.

Received for publication Jan. 30, 1945.

TECHNIQUE

In performing an intercostal nerve block the skin is prepared and an intradermal wheal of procaine is raised over the chosen rib at its angle. The needle is then introduced perpendicularly through the wheal until the point impinges lightly upon the outer surface of the rib. From this point, with the bevel of the needle directed superiorly, the needle is moved until the tip just clears the inferior border of the same rib. It is advanced 0.5 cm. and aspirated carefully to exclude the possibility of injecting into a blood vessel. In this location 6 c.c. of 1 per cent procaine solution are deposited. An effective block will render anesthetic only that area innervated by the somatic nerve or nerves blocked, but it is interesting to find, in addition, that it may be accompanied occasionally by immediate relief of pain in a referred area, perhaps even on the side opposite the trauma.

The paravertebral injection is best performed with the patient lying in either the lateral recumbent position or the prone position. The sites for injection are marked on the skin, opposite the spinous processes and approximately 4 cm. lateral to the midline on the affected side. These points lie over the transverse processes. For the best results one or two additional ganglia cephalad to the area of involvement should be incorporated in the block. At least three-inch needles are required. Each needle is introduced perpendicularly through the skin to a point where contact is made with the dorsal surface of the transverse process. With the bevel of the needle directed medially, the needle is redirected anteromedially and passed just inferior (or superior) to the transverse process and advanced until the point of the needle impinges lightly on the body of the vertebra. At this point the needle tip lies in a location such that, when an injection of 6 c.c. of procaine solution is made, there will be sufficient infiltration to block the paravertebral sympathetic chain as well as the adjacent intercostal nerves. If, during the insertion of the needle, blood or spinal fluid appears, the needle is withdrawn and reinserted in a slightly altered direction. The needle is aspirated carefully in two planes to exclude the possibility that the needle tip may be lying in either a blood vessel, a prolongation of the subarachnoid space, or the pleural cavity. With a negative aspiration, 6 c.c. of 1 per cent procaine solution are injected. If the solution is deposited properly, relief from pain is realized immediately, time being required only for infiltration of the solution.

CASE REPORTS

CASE 1.—An American soldier, aged 28 years, was wounded in action March 2, 1944, at 12:15 A.M., when a building in which he was sleeping was bombed. The patient suffered contusions of the left side of the chest with simple fractures of the fifth to the tenth ribs posteriorly and the second and third ribs anteriorly. Following injury the patient was dyspneic and could only breathe with "short gasps." The pain in the chest was excruciating, especially when the patient was moved. The left side of the chest was motionless. The fifth to the eleventh left intercostal nerves were blocked with 1 per cent procaine, March 2, 1944, at 9:15 A.M. The pain was immediately relieved. Dyspnea disappeared and the patient became comfortable. The block was not repeated because the pain did not return.

CASE 2.—An American soldier, aged 21 years, was injured in an automobile accident on Sept. 27, 1943. He complained of hemoptysis during the first day with persistent pain on coughing and deep breathing. He was transferred to the chest service on Sept. 29, 1943, dyspneic and uncomfortable, with pain in the left lower chest. There was no external evidence of injury. The percussion note was resonant bilaterally, but there were diffuse wheezes and rhonchi throughout both lungs. The patient was encouraged to cough but was

unable to because of the severe pain. Pressure over the left seventh, eighth, and ninth ribs in the anterior axillary line elicited exquisite tenderness, but roentgenograms of the chest revealed no fractures. In spite of the fact that no fractured ribs could be demonstrated, an intercostal block of the left lower six ribs was performed. This procedure relieved the pain completely, and the patient coughed up several tablespoonfuls of thick, white, tenacious sputum. Approximately two hours after the nerve block physical examination of the lungs revealed normal findings. No subsequent block was necessary.

CASE 3.—A 27-year-old soldier sustained a "sucking" wound of the right posterior thoracic wall, Sept. 13, 1943. The wound was débrided and closed within twenty-four hours. A tension hemopneumothorax developed which was treated by means of an indwelling needle with attached flutter valve. On admission to the chest center, Sept. 18, 1943, the patient was slightly dyspneic but had no acute respiratory distress. Roentgenogram revealed the right lung to be 30 per cent collapsed. About twelve hours after admission, pain developed in the right side of the chest which grew progressively worse. Very shortly the patient showed marked respiratory difficulty. He began to perspire, became moderately cyanotic, and had a "wet," painful cough productive of only small amounts of tenacious mucoid sputum. Loud, coarse rhonchi were heard over both lung fields posteriorly. The immediate treatment consisted of right-sided intercostal nerve block from the fifth to tenth ribs, inclusive, followed by a thoracentesis. Twelve hundred cubic centimeters of bloody fluid and air were removed from the pleural cavity. Thus, pain and the splinting effect of intrapleural fluid were simultaneously alleviated. Almost immediately, the patient was able to breathe more deeply and to cough effectively. In a short time the patient stopped perspiring and the bronchial râles disappeared. Within the following five hours he raised six ounces of tenacious, mucoid sputum. The next day a roentgenogram showed remarkable clearing of the right lung. The patient convalesced uneventfully.

CASE 4.—A 22-year-old American soldier sustained a severe, perforating, shell-fragment wound of the left thorax, Sept. 22, 1943. On admission to the chest service five days later, there were persisting hemophysis, dyspnea, and painful cough. There was a large gaping wound of entrance along the upper mesial border of the left scapula. Showers of coarse bronchial râles were heard over the entire left lung. Roentgenograms revealed compound, comminuted fractures of the scapula and the second and third ribs posteriorly. Relief of pain and improvement of bronchopulmonary drainage were urgently indicated, but the position of the large gaping wound necessitated a paravertebral block of the first, second, and third dorsal segments. The pain in the thoracic wall was immediately relieved. The patient's dyspnea and râles disappeared as cough and expectoration became effective. He was able to sleep well without sedation. Two days later recurrence of respiratory distress was indicated by increasing thoracic wall pain, wheezing respirations, dyspnea, and "wet" cough. The paravertebral block was repeated with the same beneficial effects. Convalescence, in this patient, also was uneventful.

COMMENTS

The indications for therapeutic nerve block in thoracic injuries have been discussed. The simplicity and efficacy of this therapy, whenever pain of any degree follows thoracic trauma, merit its more general application. The employment of both the intercostal nerve block and the paravertebral nerve block has been discussed.

This therapy has enabled us to dispense with all chest strapping except in patients with a severe degree of "flail" chest. Here, the continuance of paradoxical motion may necessitate immobilization by means of adhesive strips. Likewise, the use of morphine sulfate for the relief of pain is no longer necessary in these cases. The depression of the cough reflex and the diminution of respirations which so frequently occur with morphine need no longer complicate the already impaired respiratory efficiency seen in chest trauma.

Techniques have been described for blocking the pain stimuli originating either in contused areas or in fractured ribs. Cases have been cited.

No distinction has been made between the paravertebral dorsal nerve block and the paravertebral sympathetic nerve block because it is felt, that, with the injection of 6 c.c. of procaine solution, there is infiltration into both areas.

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RECOVERY FROM HEMOLYTIC STAPHYLOCOCCUS AUREUS BACTEREMIA ATTRIBUTED TO PENICILLIN THERAPY

Case Report

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THE literature abounds with references dealing with all phases of penicillin therapy of various infectious processes. A significant study on the effect of penicillin on surgical infections has been reported by Lyons.[1] The curative effects of penicillin in cases of staphylococcic infections with sepsis have been of particular significance since, as is well known, no previous mode of therapy has been of any real value in cutting down the high mortality of staphylococcic bacteremia. Evidence is rapidly accumulating that in penicillin an agent has been developed, which will prove consistently effective in combating the heretofore lethal staphylococcus. Florey and Florey[2] have reported ten patients with severe staphylococcal infection, three with staphylococcal septicemia, all of whom recovered following the administration of penicillin. Blake and Craige[3] have reported two patients with staphylococcic suppurative pulmonary disease that were brought to the state of clinical cure by the administration of penicillin.

It is felt that the following case of hemolytic *Staphylococcus aureus* septicemia resulting from a thoracic surgical wound infection, and presenting significant pleural and pulmonary complications, with clinical cure by the use of penicillin presents enough features of interest to warrant reporting.

CASE REPORT

This patient was wounded in the right chest by a high explosive fragment on July 19, 1943. The wound was débrided shortly thereafter and he was evacuated to the rear, entering this service on July 24, 1943. Examination disclosed a serrated missile, measuring 3 by 4 cm., lying in the right pleural cavity, and a moderate degree of intrapulmonary pathology and a moderate, clotted hemothorax on the right. During the subsequent five days, the intrapulmonary lesions cleared to a considerable extent and on July 29, 1943, a right posterolateral thoracotomy was done. The intrapleural foreign body was removed and the organizing clot evacuated. No sulfanilamide was placed in the chest or in the wound, and the

Received for publication, Jan. 24, 1945.

incision was closed with water seal catheter drainage. The patient withstood the procedure very well, and the first few postoperative days were uneventful.

On Aug. 5, 1943, seven days after operation, the temperature rose to 101° F. and the patient began to complain of pain in the wound, which revealed some induration. Hot wet dressings were applied; the wound was opened in its central portion and a culture was taken, although no pus was found at this time. The culture failed to grow any organisms. The following day, both the swelling and induration of the wound increased and the temperature remained elevated. The wound was opened further and although no frank pus was found, culture now was positive for hemolytic *Staph. aureus.* During the course of the next few days, the temperature was consistently lower and the wound induration subsided somewhat. On August 10, when frank suppuration first appeared, additional drains were placed in the wound. Nevertheless, the next day the temperature became more elevated. Under sodium pentothal intravenous anesthesia, the wound was opened widely and several additional pockets of pus were opened and drained. Following this procedure, wet dressings were resumed, and the temperature declined.

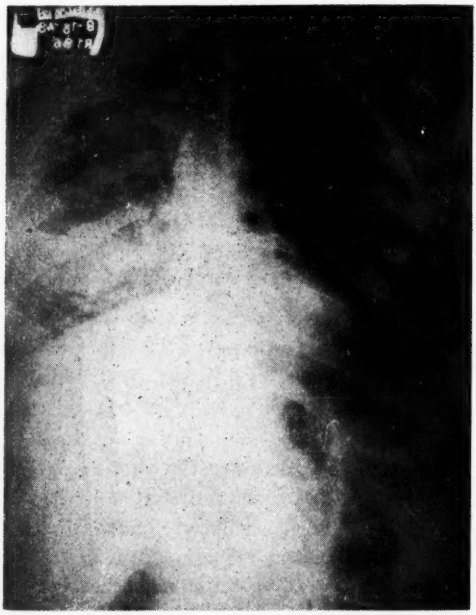


Fig. 1.—Empyema pocket in the right pleural cavity; numerous small metastatic foci in the left lung.

On August 11, the temperature again rose to 102° F. and the patient complained of abdominal pain, which was accompanied by moderate distention and definite tenderness in the left upper quadrant, suggesting splenic involvement. At the same time, it was found that he had infected emboli on the distal portions of several fingers and toes. A blood culture taken this day promptly grew out hemolytic *Staph. aureus.* Following an immediate intravenous injection of 4 Gm. of sodium sulfathiazole, he was given 1 Gm. of this drug every four hours. Daily transfusions of citrated blood were started. On the following day the temperature was still septic and the patient became very toxic. A roentgenogram (see Fig. 1) revealed an empyema on the right and multiple metastatic foci in the left lung. The empyema pocket communicated with the central part of the wound and was drained. The infected emboli of the extremities showed suppuration and were incised. At the same time new emboli appeared on several other toes and fingers as well as on the forearms.

On August 17, the sulfathiazole therapy was discontinued and penicillin therapy was started. Up to this period the patient's course was steadily downhill and his general condition was rapidly deteriorating. It was obvious that repeated blood transfusions and sulfathiazole therapy were having no effect in staying the trend toward a fatal termination.

An initial dose of 50,000 Florey units in 500 c.c. of sterile water was given intravenously, and repeated six hours later. This was followed in another six hours by 25,000

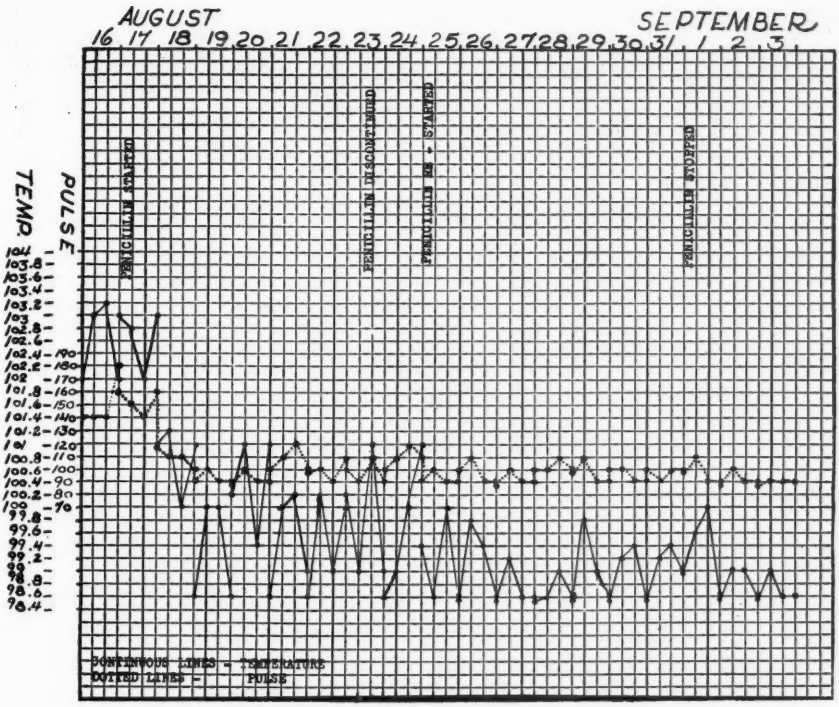
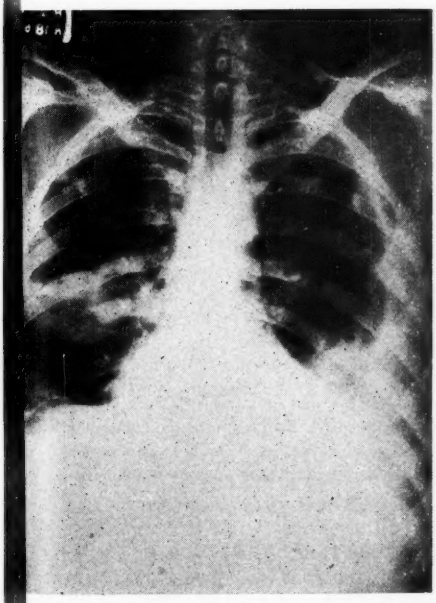


Fig. 2.—Chart showing temperature and pulse record during penicillin therapy.



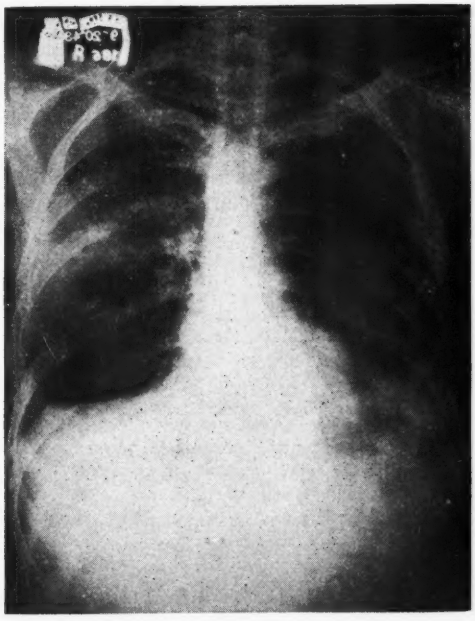


Fig. 3.—Localized empyema on the right, Aug. 23, 1943, hemorrhagic effusion in the left pleural cavity. The metastatic foci in the left lung have regressed.

Fig. 4.—There is almost complete disappearance of the bilateral pleural and pulmonary lesions.

units intravenously, and thereafter he was given 15,000 units in 3 c.c. of sterile water every three hours by the intramuscular route. There was a very immediate response as manifested by the drop in temperature and the fall in pulse rate (see Fig. 2). Within twenty-four hours the general condition was strikingly better and in forty-eight hours there was spontaneous subsidence of all the infected emboli which had appeared in the second crop. The patient continued to improve progressively.

On August 23, penicillin was temporarily discontinued because the available supply was exhausted. A roentgenogram (see Fig. 3) taken on this date revealed a localized pleural pocket on the right (which was well drained), regression of the metastatic foci in the left lung, and a moderate-sized pleural effusion on the left (found to contain thin bloody fluid). Blood cultures were still positive, although the colonies were less numerous. Penicillin therapy was resumed on August 25, but during the forty-eight hours that it was not used the temperature rose to 101° F. Upon resumption, the dosage was 15,000 units intramuscularly every three hours. On August 27, the blood culture became negative and the dosage was reduced to 10,000 units every four hours. As the blood cultures remained persistently sterile, the penicillin was discontinued on August 31.

The patient continued to maintain a steady improvement in his general condition. The wound rapidly became clean and presented healthy granulation tissue. The drain of the localized empyema cavity was gradually decreased in size and eventually removed on Sept. 6, 1943, at which time the cavity was all but obliterated. All the pulmonary parenchymal lesions disappeared completely, the hemorrhagic effusion in the left pleural cavity absorbed spontaneously, and the empyema pocket on the right became completely obliterated, as demonstrated on a roentgenogram taken on Sept. 20, 1943 (see Fig. 4). He was discharged to the rear on Sept. 25, 1943.

Follow-up studies from another hospital, two months later, stated that he was fully recovered. All wounds were said to be completely healed and roentgenograms failed to reveal any significant pathology.

DISCUSSION

The response to penicillin in this case was dramatic and conclusive. Of particular significance was the recrudescence of the infection with the temporary cessation of penicillin therapy and the prompt regression of signs and symptoms upon the resumption of the agent.

The spontaneous regression of the second crop of infected subcutaneous emboli with the institution of penicillin therapy was very striking and suggests that this same phenomenon occurred in the metastatic pulmonary foci. This resulted in their complete clearing before excavation had occurred. Equally significant was the clearing of the left-sided pleural effusion without the development of what otherwise must surely have eventuated in a staphylococcal empyema.

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ADDENDUM

This paper was not received for publication until almost one and one-half years after it was written because of delays necessitated by going through various military channels. It has now, therefore, lost much of its timeliness. It is, however, of historical importance because it was the first case of this kind in the Mediterranean theater in which penicillin treatment was successful. For that reason it is being published.

—EDITOR.

ANEURYSM OF THE SUBCLAVIAN ARTERY IN AN INFANT

Report of a Case

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MYCOTIC aneurysm is not commonly encountered. The patient reported on here was seen and operated upon in the Vanderbilt University Hospital. The case is of interest because before operation it simulated the congenital ciliated bronchial cyst of the mediastinum which may give rise to tracheal obstruction.

CASE REPORT

J. T. S., Jr., a white male, 6½ months old, was admitted to the Vanderbilt University Hospital Jan. 28, 1944. The history obtained from the mother was as follows:

The patient was a second child; pregnancy and delivery were normal. His sister, 2 years of age, was living and well. The patient's birth weight was 7 pounds, 11 ounces. His condition immediately following birth was satisfactory.

Feedings were taken well and development was normal in every way. There was no illness prior to the onset of this illness.

Six weeks before admission he developed a slight cold, followed within a few days by hoarseness and paroxysms of coughing. He was seen by the family physician, who put him in a steam tent. He improved rapidly and was considered well after one and one-half weeks. Slight hoarseness continued, however, and became more marked three weeks before admission. The mother then noted that the right eye was "droopy" and that the right pupil was smaller than the left. He was seen again by the physician, who gave him a sedative. He again improved but one and one-half weeks before admission he began to have bouts of coughing and "choking spells" which continued until admission to the hospital. During this entire time the child was given steam inhalations.

Three days before admission he was seen by a member of the Pediatric Staff of the Vanderbilt Hospital. The attacks of "choking" became more severe and were frequently associated with cyanosis. He was admitted to the hospital with a diagnosis of tracheobronchitis and possible mediastinal tumor.

Examination.—The patient was a well-developed, well-nourished infant weighing 18 pounds, 2 ounces. He appeared to be acutely ill, breathing noisely and with great difficulty. The rectal temperature was 98° F.

The skin was warm and quite moist. There was slight cyanosis which became worse when the child coughed or struggled. There was ptosis of the right upper eyelid and the right pupil was larger than the left. Both pupils reacted normally to light.

There was flaring of the alae nasi with respiration, and slight enlargement of the anterior cervical lymph nodes, but no other glandular enlargement. Some fullness was present in the suprasternal notch and the trachea was displaced somewhat toward the left side.

The superficial veins on the upper part of the anterior chest wall were prominent. Respiratory movements were labored. The rate was 38 per minute. There was marked retraction of the lower end of the sternum and of the costal margins on inspiration.

The percussion note was resonant over both lung fields. No thrill or friction rub was felt. On auscultation, many fine and coarse moist râles and rhonchi were heard over the entire thorax. No bruit was heard.

The heart was not enlarged to percussion. The point of maximum-impulse was in the fourth intercostal space. The rate was very rapid. A short blowing systolic murmur was heard at the apex but no other murmurs were heard.

Examination of the abdomen, the genitals, and the extremities revealed no abnormalities.

The red blood cell count was 4,120,000, hemoglobin 11.9 Gm. per 100 c.c., and the white blood cell count 11,850, of which 50 per cent were lymphocytes.

Roentgenogram of the chest (Fig. 1) revealed displacement of the trachea toward the left and some narrowing of the trachea at its lower end. A large, sharply circumscribed,

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Received for publication July 18, 1945.

dense shadow was present in the upper mediastinum, extending well into and partly obliterating the right upper lung field. These changes were thought to be due to a tumor, probably a cyst of the mediastinum.

Fluoroscopic examination, performed several hours after admission to the hospital, revealed essentially the same findings as are described here. No pulsation of the tumor was noted.

Course in Hospital.—Following admission to the hospital, the child was placed in an oxygen tent and his color was better for a short time. Thereafter, the respiratory efforts became progressively more labored and bouts of coughing became gradually more frequent. Cyanosis became progressively more marked.

Twenty-four hours after admission the patient was taken to the operating room. It was believed at this time that the child had a cyst of the superior mediastinum.

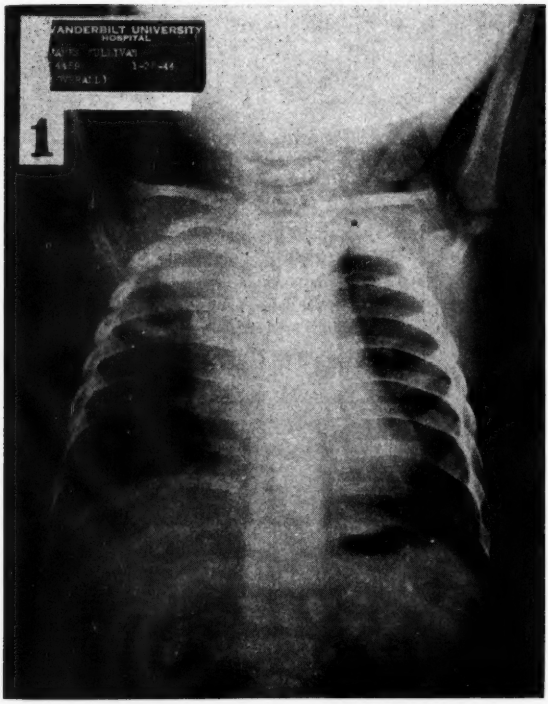

Fig. 1.—Roentgenogram of the chest.

Operation.—The anterior right side of the chest wall was anesthetized with 1.0 per cent procaine solution. Oxygen was administered by means of a tightly fitting mask.

A transverse incision was made over the right third intercostal space anteriorly and laterally. The fibers of the pectoral muscle were divided. The third and fourth costal cartilages were divided and an adequate exposure of the right pleural cavity was obtained. A large smooth tumor mass was seen to extend from the superior mediastinum downward and laterally almost to fill the upper third of the pleural cavity. It lay beneath the parietal pleura. It was very hard and tense to the touch and no pulsation could be felt. The parietal pleura was divided over the lateral aspect of the tumor and this incision was carried posteriorly. At this moment the tumor ruptured at approximately the place at which the pleura had first been divided. This rupture was spontaneous, since neither the operator's hands nor any instruments were touching the tumor at the time of its rupture. There was a great gush of bright blood from the tumor. This was controlled for a few moments by the surgeon's finger, which was placed over the point of rupture. The wall of the cyst was extremely friable, however, and the surgeon's finger gradually sank into the cavity within the tumor so that a greater defect was created in its wall and profuse bleeding occurred. A gauze pack was placed against the mass but in spite of considerable pressure, the bleeding could not be controlled. The patient died approximately three minutes following rupture of the aneurysm.

Autopsy.—Post-mortem examination revealed a large aneurysm of the right subclavian artery. The sac was a false one, being composed of friable granulation tissue and connective tissue, and also covered by the parietal pleura. The superior wall of the vessel was found to be intact, but there was a defect in the inferior wall of the artery about 1.5 cm. long. Lying in the inferomedial wall of the aneurysm was a large friable lymph node which apparently formed a part of the aneurysm wall at this area (Fig. 2).

Microscopic sections, made from various areas in the wall of the aneurysm, revealed the wall to be composed for the most part of a thin layer of fibrous connective tissue within which was a layer of granulation tissue and fibrin. Within this was a laminated shell of blood clot which was undergoing organization in its outer layer. The lymph node showed evidence of acute inflammation; polymorphonuclear neutrophiles were seen scattered through the granulation tissue which lined the sac.

There was nothing found which had the appearance of a neoplasm. It is thought that infection may have extended from the large lymph node which lay against the vessel wall to involve the wall of the artery, giving rise ultimately to its rupture and to the formation of the aneurysm.

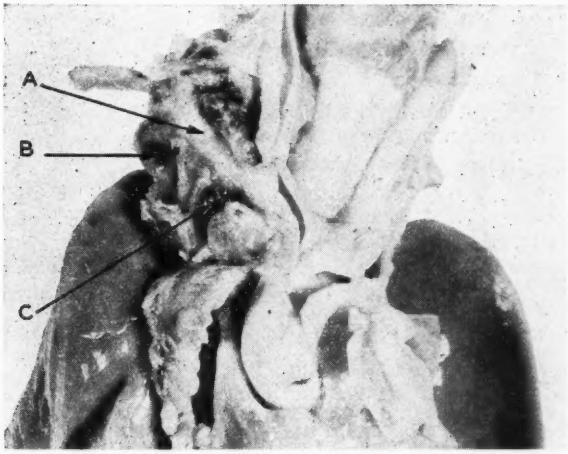

Fig. 2.—Photograph of specimen removed at autopsy. *A*, Subclavian artery; *B*, the aneurysm; *C*, large lymph node lying in the inferomedial wall of the sac.

DISCUSSION

Had it been possible to establish the diagnosis of this lesion before operation, exploration of the chest would probably have been carried out anyway. The result might have been the same, therefore. It should be emphasized that there was no demonstrable pulsation of the mass. This was specifically sought prior to division of the parietal pleura. It also should be noted that only the parietal pleura overlying the tumor was incised. It seems, therefore, that this structure alone was responsible for preventing spontaneous rupture of the soft wall of the false aneurysm before operation. Had a needle been placed into the lumen of the sac when the tumor was first exposed, rupture at the point of puncture might have occurred.

The only approach to the lesion which could conceivably have enabled the surgeon to gain control of the blood vessels which were connected with the sac is through the anterior neck. Division and elevation of the clavicle or median sternotomy might have permitted ligation of the innominate artery near the aorta and of the subclavian and common carotid arteries distal to the aneurysm. The proximity of the sac to the origin of the common carotid artery makes it doubtful that ligation of the subclavian artery proximal to the sac could have been accomplished.

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1945

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NOTICE

The American Association for Thoracic Surgery will hold its next meeting in Detroit, May 29, 30, and 31, 1946, with headquarters at the Statler Hotel. Dr. Edward J. O'Brien is the chairman of the local committee on arrangements, but requests for reservations should be sent to the hotel.

Applications for places on the program must be submitted before March 1, 1946, to the secretary, Dr. Richard H. Meade, Jr. It is requested that abstracts of the proposed papers be submitted in triplicate. The members of the program committee are: Dr. Claude S. Beck, chairman, Dr. I. A. Bigger, Dr. Evarts A. Graham, Dr. Richard H. Meade, Jr., and Dr. Edward J. O'Brien.

Nominations for membership must be sent before Feb. 1, 1946, to Dr. Ethan F. Butler, 956 W. Water St., Elmira, New York, chairman of the membership committee.

The Rose Lampert Graff Prize for the best paper submitted for the meeting will be offered again this year. The committee to make this award is composed of Dr. Frank B. Berry, chairman, Dr. Richmond L. Moore, Dr. Max Pinner, and Dr. J. J. Singer. Anyone desiring to enter this competition must submit abstracts and the completed papers. The papers will be considered first by the program committee, and then forwarded to the prize committee.

Announcement

Due to conditions beyond the control of the editors and publishers, several issues of the JOURNAL are printed on an inferior grade of paper. Just as soon as the standard good grade of paper is available, its use will be resumed.

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